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FIG. 1.



Hand affected with dry gangrene. (See also Fig. 2, page 92.)

# INTERNATIONAL CLINICS

## A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND  
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ON

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PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
OTOLOGY, RHINOLOGY, LARYNGOLOGY,  
HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD

EDITED BY

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# Diagnosis and Treatment

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## PROFESSOR CIESIELSKI'S THEORY OF SEX DETERMINATION

BY S. W. CARRUTHERS, M.D. (EDIN.)  
Norwood, England

---

THEORIES of sex have been numerous, but very few have been long maintained in any rigid or simple form. The factors adduced in the past have been very varied in character—from those that are entirely extraneous, such as the phases of the moon, to intimate and molecular ones, such as the nitrogen content of the gametes. The tendency during the last decade was to deal with general theories of anabolic or catabolic diathesis rather than to lay stress on any individual factor. The very latest investigations, however, point to extremely definite factors in the constitution of the germ-cell and the sperm-cell, and are part of the battle-ground of the mendelians and the anti-mendelians at the present moment. Theories which suppose that sex can be influenced during the development of the individual are now almost entirely discredited, and those which postulate that the sex is irrevocably fixed at the moment of impregnation hold the field.

Professor Ciesielski's theory is not dependent on any anatomical or histological investigations, but is based entirely on practical breeding experiments, chiefly in plants. It appeals to the inquirer in two or three respects, and, as it seemed worthy of a wide diffusion in English form, I obtained his sanction to issue a translation. Three points seemed to appeal specially for a favorable consideration of the hypothesis.

First, its publication was delayed for many years after the author had convinced himself of its truth. In this it differs from some widely-trumpeted theories published promptly upon formulation and speedily remoulded into almost unrecognizable shape.

Secondly, the investigator has himself tested his view experimentally to his own satisfaction in plants, in various animals, and in man. Many other theories have been admitted by their authors to apply only to some parts of the animal or vegetable kingdom.

Thirdly, the theory is of the utmost simplicity. It depends on one factor only, and that a definitely measurable one. That this

adds to the probability of its truth one would hesitate to say, but it does not necessarily lessen the probability: and the simplicity renders the investigation of its validity very easy.

One must add, also, that Professor Ciesielski has expounded his views in a pamphlet written in Latin, a translation of which has been prepared and is here published with his consent. The original paper is a model of directness and brevity. He enters into no theoretical discussions, but relates both concisely and accurately his own observations.

It is true that the factor put forward by Ciesielski, namely, the age of the male gamete, has been mentioned by other investigators, notably by Düsing, but usually as a secondary or concomitant factor in a problem so complex that its practical working out by the breeder was little more than a lottery. Professor Ciesielski claims that no one before him had proved this to be the all-important factor, and that he has thus provided a simple general rule, which can be practically used in breeding plants, animals, and human beings.

It would not become me to enter into a critical discussion of the theory; but it is of some importance to know whether it is in diametrical opposition to the results of other reliable observers or not. Cases of long storing up of the male elements in the body of the female (as in many species of bats, bees, ants, and earthworms), while they, of course, violate the 24-hour limit, do not disprove the general thesis that older semen produces females. It is evident that in these cases the environment is a special one provided by Nature to preserve the sperm-cells in unchanged activity, and cannot be paralleled by cases where no such provision exists.

Taking the theory which considers catabolism as the essence of maleness, both in the spermatozoön and in the resulting individual (soma), and anabolism as the essence of femaleness, one has only to suppose that the catabolic tendency of the sperm-cell and the anabolic tendency of the germ-cell weaken with age; then the union of a "stale" sperm with a fresh ovum would leave a marked excess of anabolic tendency and produce a female. This, it is true, does not leave the sperm-cell the sole factor, but, at any rate, it is not in direct opposition to Ciesielski's theory.

The newest and very definite observations of Professor Edmund B. Wilson as to the distribution of the chromosomes in the sperm-cell must not be overlooked in this matter. So far as Professor



Wilson's observations go, femaleness depends on impregnation by an X-spermatozoön and maleness on fertilization by a Y-spermatozoön. He quotes Morgan and von Baehr to the effect that in *Phylloxera* and *Aphis* "only the X-class, or female-producing spermatozoa, come to maturity, while the Y-class, or male-producing spermatozoa, are abortive." (Hence, of course, the fact that all fertilized eggs of these animals produce female individuals.) Now, if this extreme case of abortion of Y-spermatozoa can occur, it is not unreasonable to suppose that in other species the Y-spermatozoa might be shorter-lived than their X companions, a hypothesis which would to some extent fit in with Professor Ciesielski's results. It would explain why only females should result from stale sperm, though not why only males should result from fresh sperm.

Ciesielski's law is, therefore, at least worth serious practical investigation, and is not a difficult one to test. As regards human beings, the circumstances of married life are such that it is rarely possible to ascertain the conditions of impregnation unless the theory is explained beforehand and a deliberate experiment planned. Several medical men have written to me about this matter since I called attention to Professor Ciesielski's paper (*Lancet*, 1912, i, 260); they have kindly promised to communicate to me the results of their experiments. I shall be most grateful if any reader of this article who investigates the matter himself or in his patients by the method of accurately-planned experiment would do me the favor of communicating the details and the result. If sufficient reports arrive to justify writing another paper, all who thus help will receive due credit. I have come across only one case in which really reliable information as to the conditions of impregnation was available: and here Ciesielski's law held good in regard to all four children.

As regards animals, I have been provided with some facts observed and noted through the kindness of Mr. A. Cornish-Bowden, M.R.C.V.S., a dog-breeder of note, whose accuracy and scientific spirit are well known. From his own general experience he was strongly disinclined to believe Professor Ciesielski's theory; but his observations are absolutely impartial and his notes unprejudiced. The following are his cases:

1. Fox-terrier bitch, mated on February 15 and February 17. Dog not used previously since February 9. Result, 3 bitch pups, 1 dog.

2. Fox-terrier bitch mated on February 21 and 22 (after 16 or 17 hours' interval). Dog not used previously for fourteen days. Result, 4 dogs, 1 bitch.

3. Great Dane bitch served March 9 and 11. Dog not used for some weeks previously. Result, 6 bitches, 3 dogs.

4. Pekingese bitch mated March 11 and 18. Dog never used before. Pups born May 19, to *second* mating. Result, 1 dog, 1 bitch.

These few experiments are, on the whole, unfavorable to Professor Ciesielski's theory; but they are inconclusive. Professor Ciesielski has apparently made the time-limit too strict—at any rate, for dogs. According to his 24-hour limit, no really fresh spermatozoa were available in Cases 1 and 3, and therefore the pups ought all to have been bitches, whereas only 75 per cent. in one case and 66 per cent. in the other were bitches. But if one were to admit a 48-hour time-limit, these cases would then come under Ciesielski's rule. It is, of course, quite possible that in different animals the rate of aging of the sperm is not identical. Case 2 is in strict accordance with the law; but Case 4 seems in direct contravention of it, as the sperm-cells must have been stale on each occasion.

The view is, of course, possible that when the seminal vesicles (or the ducts of the testis) have been emptied as the result of a coitus, *all* the spermatozoa in them thereafter are fresh, and that for 24 (or 48) hours thereafter *only* males will be procreated. Later the semen will be stale: but not of necessity *all* of it. A small daily addition of spermatozoa just mature may be made from the testes. These fresh spermatozoa will, of course, form a steadily-diminishing proportion of the whole semen, and the chance of impregnation by a fresh spermatozoön will get rapidly less; especially if, as is likely, the daily addition of semen decreases as the vesicles become filled. The practical result in such an event would be that a single coitus within 24 hours after another would infallibly procreate males, while a single coitus 7 or 8 days after another would almost certainly procreate females, though now and again a stray male might occur. One must, however, frankly admit that if Professor Ciesielski's results are to be explained on the Wright chromosome theory, then the hypothesis of this paragraph cannot be maintained.

Mr. Cornish-Bowden is kindly making, at my request, a couple of experiments with only a single service of the bitch; the results should be conclusive.

# HOW IT HAPPENS THAT THE OFFSPRING OF PLANTS, ANIMALS, AND MEN IS SOMETIMES MALE, SOMETIMES FEMALE

BY PROFESSOR THEOPHILUS CIESIELSKI

---

FROM the earliest times men have been eager to solve the riddle: how does it come about that the offspring receives by heredity the nature of its parents and of its ancestors, and what factor determines sex of the offspring? I shall give here only a few examples dealing with the solution of the second problem.

Osiander of Gettinga asserted that if a woman conceived while the moon was waxing she would bear a son, and a daughter, if she conceived while it was waning. Saury was of opinion that the sex of the offspring would be that of the parent whose energy was the greater. Henke held that the right testicle and the right ovary contain male spermatozoa and ova, the left female ones, and laid down rules for sexual intercourse according to this theory.

Later inquirers have been no more fortunate in discovering the solution of the problem. Thus Thury considered that the younger ova produced females when fecundated, and the older ones males; and Albin, Flourens, Upjohn, and others agreed with him. Coste and Gerbe, however, proved that Thury's theory did not always hold good. Similarly Fiquet, Telais, and Janke held that richer or poorer nutrition was of great importance in the production of sex, and particularly affirmed that the male sex is procreated by worn-out males; but Martegoute attacked this view. Some, like Hofacker, Bertillon, Sadler, and Morel, along with many others, even proved by examples from life that the age of the parent was of importance in the production of sex—some taking the absolute ages, others the relative ages of the parents. Ahlfeld, Schramm, and Franke opposed this view. Thereafter Ploss came to the conclusion, from statistical considerations, that plenty and abundant harvests favored the production of females; but Wappäus objected to this theory. Hampe, Quetelet, Corradi, Girou, and Zenker tried to prove that the condition

of the parents and the time of year were weighty factors in the production of sex.

Finally, Düsing came to a conclusion which he explained in his work entitled, "*Die Regulierung des Geschlechtsverhältnisses bei der Vermehrung der Menschen, Tiere, und Pflanzen*" (Jena, 1884). He considered that Nature herself strove to reach and maintain a certain equality of numbers between male and female offspring, both in animals (including man) and in plants. For, he maintained, if at any time males predominate in number, then the offspring is chiefly female, whereas if females predominate, males are born. He held that the cause of this condition consists chiefly in the age of the ovum, asserting that when *recent* ova are fertilized (this, of course, happening when it is easy for a female to come across a male, *i.e.*, when males are numerous) then the fresh ovum itself produces a female; on the other hand, if males are scarce, and *older* ova are fertilized, then they produce males. Düsing, then, based his theory of the determination of sex mainly on the age of the ovum, but he allowed an influence, up to a certain point, to the spermatozoön, and also to the nutrition of the fœtus after conception.

Others, such as Landois and Treat, have tried to prove that in caterpillars and butterflies a generous diet produces females, and a scanty one males. Cuénot, however, showed this idea to be false, and insisted that the sex of the ovum is already determined before conception. Many investigators are of the same opinion even now, relying on the fact that if twins are derived from a single ovum (that is, have a common chorion) then they are always of the same sex, but if they arise from two ova (as in the case of normally polyembryonate animals), then they may be either homosexual or heterosexual. In 1898 Schenck declared that if a woman had been very generously nourished she would bear a boy; but in 1900, having changed his opinion, he argued, in his book entitled "*Lehrbuch der Geschlechtsbestimmung*," that a woman would bear a boy if she were scantily nourished, and if she took, in addition, a medicine prepared by him.

The opinion of botanists as regards the origin of sex in diœcious plants does not differ much from the above theories. Some think that external causes, at the period when the plant is sprouting from



the seed, have a great influence over the sex. Others lean to the opinion that the sexual distinction is already present in the seed itself. But in this matter neither the treatises and experiments of learned men—Knight, Gärtner, Mauz, Hoffman, Heyer, Molliard, Haberlandt, Leidhecker, Sollardo—nor even the latest investigations of Magocsy-Dietz, in 1907, have provided anything definite; so that to-day the matter of the origin of the sexes seems to be involved in complete darkness, both among the laity and among doctors and scientific men.

After studying, year by year, from 1871 onward, the question of the origin of sex in *Cannabis sativa*, I discovered, in 1878, an unchanging and settled law of Nature, in accordance with which it happens that sometimes male *Cannabis* plants and sometimes female ones are produced, so that if we keep the law clearly in view we can in very simple fashion control this enigma of Nature.

For thirty-three years I hesitated whether I ought to publish this law of Nature, for I feared that the knowledge of it would tend to lower the moral standard, but now at length I am satisfied that I ought not to delay, for it may well happen that someone else may not hesitate to make this same discovery public.

My experiments were as follows: In 1871, having been appointed Keeper of the Collections at the Breslau Botanical Gardens, I began to experiment upon *Cannabis sativa* and *Spinacia oleracea*, in the hope of finding out why sometimes male and sometimes female plants were produced. Professor Richard Göppert, at that time Director of the Gardens, gave me an area of some 20 square metres for these experiments.

I had learned that farmers believed that the more thickly *Cannabis* was sown, the more male plants would result (male plants being more valued in agriculture for their fibre). I, therefore, sowed 1000 selected seeds of *Cannabis* and the same number of *Spinacia* in rows, some at equal distances of 20 cm. apart, and others at distances of 10 cm. When the plants were mature, I took a census, and found that in the *Cannabis* which had been sown sparsely there were 486 male plants and 475 female, while in that which was thickly sown there were 465 male plants and 482 female. The other seeds either did not germinate or else died immature. The *Spinacia* plants I

did not count, for they were damaged. I learned from this experiment that the view that thick sowing favored the production of male plants was erroneous.

In 1872 I made a fresh experiment with the same *Cannabis*, sowing a thousand seeds at each quarter of the moon, in view of the common belief that the seeds of *Cannabis* sown in the first quarter and at full moon produce more male plants, while those sown in the last quarter and at new moon produce an excess of females. But this view was also shown by experiment to be groundless.

In the autumn of 1872 I was called to the chair of Botany in the University of Lwow (Lemberg); and in the early spring of 1873 I bought some land in Lyczacov Street, in Lemberg, stretching more than 200 metres in length from north to south, and sloping to the south, almost divided into two equal parts by some buildings. This plot seemed to me very suitable for carrying on my series of experiments with *Cannabis*.

To ascertain whether sunlight was, perhaps, of importance in determining sex, I sowed half my seeds of *Cannabis* on the slope facing south and the other half in the shade of the buildings; but this also failed in solving the problem, for in each locality the male and female plants were almost equal in number. In 1874 I took the seeds from plants grown in the shade and from those grown in the sun, and, dividing each into two parts, I sowed one half in a sandy soil on the slope, and the other in the lower part of the garden, well manured; but here also male and female plants occurred in similar proportions in both localities. In all four sections the male and female plants were found mingled indiscriminately. Each section occupied an area of 2 square metres; and the number of male plants varied usually between 40 and 50 per cent., sometimes one sex and sometimes the other predominating, but the female plants being in the majority.

In the same year I sowed two plots, one on the slope protected by a white linen covering, the other one the lower ground with no covering, in order to discover whether perhaps excessive drought or moisture were factors in determining sex. I watered the first plot so sparingly that in the midday hours the plants almost withered; the other I watered daily very liberally. This also was of no avail in discovering the explanation; and even when, in the following year, I sowed the seed gathered from these in different areas, no distinction

could be observed as the result of the lack or abundance of moisture.

In 1875 I constructed, in the upper part of the Lemberg Botanical Gardens (where in my predecessor's time useless herbs and woody shrubs had grown), a field for investigating the nature and life of plants. One portion of this was marked out in sixteen plots, one metre broad and eleven metres long, divided by broad furrows into ten smaller plots of one metre square. I manured this field with compost (*i.e.*, mold prepared from various decaying materials), and thereafter I dug in thoroughly a different manure in each of the sixteen plots. Plot 1 remained without special manure; 2 had guano; 3, a manure mixed from the nutrient salts which we use in experiments for artificially feeding plants in water; 4, chloride of potassium; 5, phosphate of lime; 6, phosphate of potash; 7, nitrate of potash; 8, nitrate of soda; 9, sulphate of lime; 10, sulphate of magnesia; 11, bone meal. Each of these was in the proportion of 200 kilos. per hectare. Plot 12 had human dung; 13, the dung of sheep; 14, that of pigs; 15, that of horses; and 16, that of cattle, in each case in the proportion of 21,400 kilos. per hectare.

I sowed these small plots cross-wise with the seeds of ten different plants, and among them a transverse row of sixteen small plots of *Cannabis*.<sup>1</sup> In the adult *Cannabis* plants no distinction produced by these various manures could be observed. In the unmanured area there were 45 per cent. of male plants, and in that with human dung there were 45.5 per cent.; in all the others the number of male plants varied from 48 to 52 per cent., there being a preponderance in one area of male plants, in another of female ones.

In my garden in Lyczakov Street I sowed *Cannabis*, in 1875, in three divisions more than 100 metres apart, and as soon as I could detect the sex of the plant I cut away two-thirds of the inflorescence of all the female plants, leaving the uppermost third in one division, the middle third in the second division, and the lowest third in the last division. I gathered the seeds from the different parts of the inflorescence, and then, in 1876, I sowed them separately in my garden, once more in three places similarly located. Not even then did any decided difference manifest itself in the sex of the plants derived from these different parts of the inflorescence.

Since I had now convinced myself by these experiments that in

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<sup>1</sup> The result of one such experiment was published in *Bartnik postepowy*, a monthly journal of horticulture and apiculture, which I have edited since 1875.

*Cannabis* no external cause was effective in the production of sex, and that sex-difference was determined in the seed itself, even before the plant was produced from it, I began another method of investigation, and in that same year (1876) started experiments with artificial fertilization of the flowers. As soon, then, as the sex of the plants in the three above-named divisions could be determined, I took out all male plants from the upper and lower divisions of the garden, leaving only female ones; whilst in the middle division of the garden, which was surrounded by buildings, I left only male plants. Thereafter, as soon as the female plants were mature, I fertilized them with pollen from male plants carefully cut down from the middle part of the garden, simply by shaking the male plants over them. The female plants in the upper garden I fertilized daily at sunrise, but those in the lower garden just before sunset.

The seeds collected from these two divisions I sowed again, in 1877, separately in the upper and lower portions of the garden, but in new plots, to avoid any possible fallacy from seeds of the former experiment casually and spontaneously shed in the old plots. The result was that from seeds derived from plants fertilized at sunrise I obtained 85.5 per cent. of male plants, while the seeds gathered from plants fertilized at sunset produced 92 per cent. of female plants.

In view of the success of these experiments in fertilization, I took up, in 1877, six female plants, with their roots, before they had fully produced their flowers, and transplanted them into pots: as soon as the plants were well-rooted I transferred the pots to the windows of two rooms looking south, three pots in each room. Thereafter, with a brush, I fertilized the three plants in one room with pollen which I collected from anthers just dehiscing and not yet fully open (this pollen I shall refer to as "fresh"), and the three pots in the other room I also fertilized by means of a brush, but with pollen taken in the morning and kept in paper till evening. The three plants fertilized with "fresh" pollen produced 120 seeds; the three fertilized with "stale" pollen produced 96 seeds.

In 1878 I sowed in my garden with great care the seeds of the two lots separately, at distances of 20 centimetres. The 120 seeds derived from fertilization with fresh pollen produced 112 plants, of which only six were female, all the rest being male. But in the



other 96 seeds, sprung from fertilization by stale pollen, produced 89 plants, every one of which was female. I have several times since repeated this experiment, always with a similar result.

Since the seeds from which the female plants sprang seemed to me round and darker, and the others more ovoid and mottled, I determined to investigate whether the distinction of the future sex was already inherent in carefully-selected seeds, so that from the appearance of such seeds of *Cannabis* one could foretell whether they would produce male or female plants. In 1880 I began, with Bronislaus Blocki, an able young man, as my co-worker, a series of experiments with seeds of *Cannabis*, varying in shape, color, specific gravity, and actual weight, but from these experiments I convinced myself that shape, color, and weight of seed have no relation to the sex of the plant produced.<sup>2</sup> Only seeds which had both of the aforesaid ovoid shape and mottled appearance produced 57 per cent. of male plants, while the number of male plants from the other classes of seeds varied, as a rule, from 40 to 50 per cent.<sup>3</sup>

Character of seeds.	Male Plants.	Female Plants.	Males, Per cent.	Females, Per cent.
Ovoid .....	146	114	56.15	43.85
Round .....	152	192	44.18	55.82
Pale green .....	88	76	53.65	46.35
Blackish .....	106	132	44.53	55.47
Mottled .....	174	176	49.75	50.25
Ovoid and mottled .....	194	130	59.88	40.12
Under average weight .....	116	134	46.04	53.06
Over average weight .....	146	214	40.56	59.44
Sinking	152 128 84 128	200 96 74 163	43.18 57.14 53.16 44.00	56.82 42.86 46.84 56.00
Suspended				
Floating				
} in mixture of alcohol and water {				
Unselected .....				

But not even from this experiment can any noteworthy conclusion, based on the characters of the seeds of *Cannabis*, be drawn.

Having reached a satisfactory result in plants, I was desirous of investigating the problem in animals also. In 1878, therefore,

<sup>2</sup> I have not undertaken experiments with other diœcious plants, because it is not unusual in them for staminal flowers to occur in female plants, an occurrence which I have only very occasionally found in my long series of experiments with *Cannabis*.

<sup>3</sup> To some little extent this experiment turned out successfully, as is shown by the following figures. Of each class given below, I sowed 400 seeds of *Cannabis*, 15 Cm. apart.

I carried out two experiments with rabbits, with successful results, and thereafter, in 1879 and 1880, I carried out a large number of experiments, both in rabbits and in dogs, which corroborated the constant law of Nature that I had discovered.

Out of the large series of experiments I here report only two of the most convincing. For the first, I took three rabbits, a black doe, a white buck, and a tawny buck. The black doe was served first by the white buck a single time, then a single time by the tawny buck, and lastly within eighteen hours was again served a single time by the original white buck. The result was as follows: a litter of five, of which one was a white female, and another a black female with white spots (both from the white buck as sire), then a black female, and a tawny female with black spots (from the tawny buck as sire), and lastly a white male with black spots (from the second service with the same white buck). In others of my experiments I did not get such accurate color results.

Quite as remarkable was the result of coupling a white bitch with prick ears, with a tan half-bred dachshund and a white kettenhund. The bitch was first served by the dachshund, then on the same day by the kettenhund, and on the next morning, within nineteen hours, by the original dachshund. She had three pups—a tan bitch pup by the dachshund, a white bitch pup by the kettenhund, and a parti-colored dog pup by the dachshund at the second service. Color is much less often prevalent in dogs than in rabbits.

In general I have observed and noted that a doe rabbit served once only by a buck which had not had connection for some days produced a litter of two or three females: but if a buck was used which had had connection the day before, then she would bear two or three males. Similarly among dogs, there is usually a litter of one or two pups from a single coitus: these are bitch pups, if the dog which has served the bitch has not had coitus for some time before. On the other hand, there are one or two dog pups if the dog which has served the dam has served another less than twenty-four hours before.

I have also proved the truth of this law in horses, for I had the opportunity, in 1880, as representative of the Town Council of Lemberg, on the Committee of Count Skarbek's Orphanage, of examining the records of the station for stallions in Mikolajow Drohowyze. By extracting from the records certain particulars

relating to the mares of the neighboring farmers which had been served, I ascertained later that the mares served by a stallion which had not served a mare for several days before produced female foals, and those served by stallions which had had coitus the day before always bore male foals.

Wishing to prove this law of Nature experimentally with the greatest possible accuracy, I bought a bull in 1897 from a half-bred herd of Simmental cattle, from a reliable man in Kabarowce, and let it for the use of farmers at my country place at Chorosciec, where I had the services of a thoroughly careful, diligent, and accurate man, the manager of my apiary.

In the years 1897 and 1898 he recorded 156 successful instances of service of cows by this bull; and when I had compared the sex of the offspring with the conditions of the service by the bull, I found that only twice did the records not agree with the law that I had laid down. I am convinced, moreover, that these two do not disprove the law, but that some mistake has crept into the records of the conditions of service.

Finally, I must not omit to record that during the last thirty-three years I have not lacked frequent opportunities of proving the law in man also, for I have been consulted by friends, relations, and acquaintances, have given appropriate instructions, and whenever these instructions have been carefully followed they have brought about successful results.

So it comes about that I can now without hesitation assert that sex of the offspring depends on the conditions of fecundation, and that this holds good in the same way without distinction in plants, animals, and men. In plants fresh pollen produces male seeds, but the effect of stale pollen is to produce female seeds. In similar fashion in animals and in men, the sex of the offspring is determined by the spermatozoa only. For fresh spermatozoa (that is, those derived from a discharge of semen within the space of 24 hours after a previous coitus) produce male offspring, but stale spermatozoa (emitted after a longer period than one day from the preceding coitus) produce female offspring.

A fact which harmonizes with this experiment is that fresh human spermatozoa move at a speed of about 0.16 mm. per second, whereas the movements of stale spermatozoa fall off, after a few days,

to a speed of only 0.04 mm. per second. But I have so far failed to find any alteration by age in the chromatine of the nuclei either of pollen grains in plants or of spermatozoa in animals.

Anyone can easily and in a short time test the law which I have elicited, if he will go through the records of horse-breeding or cattle-breeding stations where the details of service are accurately and promptly recorded. By employing dogs or rabbits the law can be tested within a few weeks. The same law holds good in bees. The queen bee is only once fertilized in her whole life by the drone. The spermatozoa of the drone, immitted into her vagina, are stored in the vesicula fructuaria, and issue thence in the act of egg-laying. These spermatozoa, therefore, are all stale, and thus, from every egg laid and fertilized by the queen, female bees alone are produced. Bees are, however, endowed with a power of producing males by parthogenesis, in such fashion that they can lay unfertilized eggs, from which males alone are produced ("arrhenotokia"). Such eggs produced parthenogenetically, and forming male bees, can be laid not only by the queen, but also by a worker bee, whose genitalia are so atrophied that she could not possibly be fertilized by the drone.

This law, moreover, explains quite simply the remarkable constancy of Nature exhibited in statistics of population, by which the proportion of the sexes is maintained in animals. For the more that males predominate over females in any species, the less frequently do they get the opportunity of fertilizing the females. It thus happens that by immitting stale spermatozoa they produce females. On the other hand, the rarer the males and the more numerous the females, the more frequently are opportunities afforded to the males for copulation, and therefore the more fresh the spermatozoa, so that male offspring are produced.

Lastly, the law explains also the fact that in periods of prolonged war it is chiefly boys that are born, and the fact that when old men wed young women the offspring are chiefly boys, whereas young men with older women produce chiefly girls; and, finally, that tuberculous fathers beget a preponderatingly male offspring. It also follows necessarily that twins arising from one ovum (*i.e.*, with one chorion) and from a single conception are both of the same sex; whereas twins which have sprung from two ova are of the same sex if the ova were fertilized at the same time, or at similar periods of fecundation; but



that they are of different sex if the fertilization were not at the same time and were at dissimilar periods of fecundation.

In the Eleventh Congress of Polish Medical Men and Naturalists, at which I first made this investigation public, I was met with the objection of Professors Nussbaum, Siedlecki, and Godlewski, Jr., that I had made no new discovery, because the facts which I set forth had been already recorded, and especially because the learned Düsing had already brought forward the same theory. I quote here the words of Düsing in explanation of his chief theory, on page 281 of the book aforementioned: "It has been shown that the individuality of the mother has an influence on the sex of the offspring. This reaches its expression through the qualitative peculiarities of the egg, in which, even before fertilization, there must be a tendency to develop into one or other sex; *e.g.*, young ova tend toward the female sex, older ones, on the other hand, toward the male sex.

"The facts show further that the individuality of the father, *i.e.*, the qualitative constitution of the semen, exercises an effect on the production of sex. By it the above-named tendency of the unfertilized ovum can be altered in the act of fertilization. The forces inherent in the personality of the father and of the mother, which reach their expression by means of the quality of the sperm and the ovum, may also work with varying strength in one or the other direction. They unite together, then, to a resultant whose outcome impresses on the ovum its preliminary tendency of sex formation."

On page 282 he writes on the same point: "At fertilization the sex of the embryo is, however, not yet definitely decided. We know that the last factor to develop, that of nutrition, can still make its influence felt for a time. The influence of the mother's nourishment on the production of sex can make itself felt in mankind for three months. During that period, in the case of twins, there is no encroachment on nutrition. In the case of triplets, however, even earlier than this there manifests itself a considerable competition for nourishment, and so more boys are found among triplets than among twins."

On page 49 of the same book he writes as follows: "A greater age of the ovum at fertilization constantly produces a predominance of male births; and, on the contrary, a smaller age produces an excess of female individuals."

From all these passages it is evident then that Düsing attributes to the ova, to the spermatozoa, and also to nutrition after conception, to each its own influence in determining the sex of the fœtus; but nowhere does he attribute the determination of sex to the *single* factor which I have elicited, namely, the freshness or the staleness (beyond one day from the last coitus) of the spermatozoa.

Lastly, the way in which Düsing understood the problem of the origin of sex is best indicated by his experiment with *Cavia cobaya*, described in pages 312 to 317 of the same book. For he divided his animals (almost 90 in number) into two classes; in one the females were absolutely in excess, and in the other the males. By this method he wished to demonstrate to the eye that his opinion as to the law of the equation of the sexes in nature is true; but in this aim he has not succeeded. He did not, as a fact, undertake any experiment to demonstrate the factors on which the origin of sex, male and female, generally depends.

Among botanists, Hoffman made some experiments in the artificial fertilization of plants—for example, *Mercurialis annua*, wishing to test Thury's theory—but the result did not justify his attempt. He fertilized with old pollen (kept from September of the previous year) both old and fresh ovules, and similarly, with recent pollen (he does not state how recent), both old and fresh ovules. Yet he deduced from the experiment this final theory: "It follows, therefore, either (*a*) that neither the earliest possible fertilization nor the latest possible fertilization has any influence at all on the sex; or else (*b*) that the fallacies are so important that they completely mask the true state of the case." (*Botanische Zeitung*, 1871, 103.)

## THE TONUS OF THE VAGUS

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THE citation of simple manœuvres to attain puissant results does not impugn scientific medicine; on the contrary, it demonstrates the paths of least resistance in combating reflex phenomena. Much in physiotherapy has justly been discredited, owing to exaggerated statements emanating from incompetent sources. Cures mean nothing to the scientist, and in the application of my methods I have never been influenced by empiricism alone, and the elicitation of my reflexes to combat disease may easily be demonstrated by anybody reasonably skilled in physical diagnosis. The subject of vagus tone has occupied my attention for years and it is only recently that anything approaching the confirmation of my investigations has appeared. In a monograph,<sup>1</sup> which is largely hypothetic, emanating from the von Noorden clinic, an endeavor has been made to demonstrate the relation of the tone of the vagus to other diseases. Inasmuch as there is no evidence in this monograph to recognize the tone of the vagus by its effects on the visceral reflexes, the discussion is necessarily theoretic.

Before studying the subject of vagus tone, certain facts, anatomic and physiologic, must be recapitulated concerning the pneumogastric nerve. First of all, something must be said of the nervous system, which is divided into the cerebrospinal and sympathetic. The former consists of the brain, spinal cord, cranial and spinal nerves. It supplies the special senses and the voluntary muscles. The sympathetic presides over the visceral movements, controls the phenomena of secretion and influences the calibre of the blood-vessels.

Anatomically both systems are with difficulty differentiated, but this difficulty is surmounted by the use of nicotine. If the sympathetic fibres are painted with nicotine, their functions are inhibited, whereas the same agent is without effect on fibres of the cerebrospinal system. The sympathetic system is composed of fibres which according to their origin may be divided into cranial, bulbar, and sacral.

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<sup>1</sup> Die Vagatonie: Eppinger und Hess. Berlin, 1910.

1. *Cranial division*.—This is composed essentially of fibres which pass to the eye through the oculomotor nerve.

2. *Bulbar division*.—The fibres of this division pass through the facial and glossopharyngeal nerves and innervate the glands and blood-vessels of the head. The main nerve of this division is the *vagus*, which is the chief nerve of the viscera.

3. *Sacral division*.—This innervates the arteries of rectum, anus, external generative organs, walls of descending colon to end of gut, walls of urethra and bladder and muscle of external generative organs.

*Further differentiation of the sympathetic*.—All the fibres of this system which run into the gangliated cords of the sympathetic are known as sympathetic fibres, whereas the others, called *autonomic*, represent essentially the extended *vagus*.

These two sets of fibres are physiologically in antagonism; the irritation of one set inhibiting the functions of the other. Each set shows a definite pharmacologic reaction equivalent to their electric stimulation.

*Adrenalin* acts exclusively on the sympathetic, whereas the autonomic fibres are stimulated by *pilocarpine*. The action of *atropine* is peculiar. It may inhibit the action of other drugs on the autonomic fibres and while its action is most powerful on the cranial division, it is practically without effect on the sacral division.

In health, the viscera are in a state of tonicity, *i.e.*, their musculature is in a more or less permanent although variable condition of contraction. The sympathetic fibres are stimulated experimentally by adrenalin (sympathicotropic action), and the tonus of these fibres in the organism is maintained by the constant secretion of adrenalin and other products (epinephrin, suprarenalin) from the adrenal bodies.

A similar internal secretion has not yet been demonstrated for maintaining the tonus of the autonomic fibres, although we know that such physiologic action can be exhibited by pilocarpine (vago-tropic action).

The pancreas has an inhibitory influence on the secretion of adrenalin and after extirpation of the pancreas, adrenalin is increased. When the adrenal secretion is augmented, the reflexes of the sympathetic fibres are increased, and conversely, diminished when the secretion is reduced.

The pharmacologic excitation cited is analogous to what occurs



when the sympathetic fibres supplying the iris are cut, *viz.*, pupillary contraction, and dilatation of the pupil, when the autonomic fibres are divided. In the norm, instillation of a drop of one per thousand solution of adrenalin into the eye has no effect on the dilator pupillæ, but if the sympathetic system is excitable, pronounced mydriasis follows the instillation.

The tenth or pneumogastric nerve (*nervus vagus*) is the longest and most extensively distributed nerve and contains motor and sensory fibres. It is *motor* for the soft palate, pharynx, larynx, bronchial muscle, heart, and abdominal organs and *sensory* for the pharynx, larynx, trachea, œsophagus, and probably the heart. There are many problems in the physiology of the vagus which have not been solved by the physiologist and in this regard much may be anticipated from clinical observations.

In my experimental and clinical work, I have concerned myself chiefly with the tonus of the vagus and clinical pictures have been evolved which are identified either with a diminution of vagus tone (*vagus-hypotonia*), or an augmentation of vagus tone (*vagus-hypertonia*). Variations in vagus tone may involve the entire nerve, or it may be confined to one or more of its individual branches (*local vagus-hypotonia*, or *hypertonia*).

Humans, like animals, show variations in vagus tone. Thus, in some animals, section of the vagus (vagotomy) will produce tachycardia, whereas, in other animals no such action is observed. The vagus is more active in middle life than in old age and least active in infancy. In some humans infinitesimal doses of atropine (which inhibit vagus impulses) will produce tachycardia, mydriasis, glycosuria, etc., whereas, in others large doses of the same drug produce scarcely any effects.

When the vagus is diminished in tone, it produces symptoms varying in the motor sphere from hypotonia to paralysis and, in the sensory sphere, from hyperæsthesia to anæsthesia. Increased tonus in the motor sphere is associated with spasms and in the sensory sphere with hyperæsthesia.

Psychic factors have an important influence on the tone of the vagus and an appreciation of this fact will explain many problems. Emotional excitement stimulates adrenal secretion, and it is evident that, when the sympathetic is stimulated, the tonicity of the vagus is reduced.

I have often been impressed with the inconsistency of our conception of hysteria as a disease in which the *will* controls the body and produces morbid changes in its functions. The fact is, the symptoms of the disease are caused by stimulation of the sympathetic system and the latter is not under the influence of the will. It is, therefore, inconsistent to ask such patients to control their symptoms by exercise of the will. Inasmuch as adrenalin acts exclusively on the sympathetic, and pilocarpine on the autonomic fibres, adrenalin will ameliorate symptoms caused by augmented vagus-tonus, whereas pilocarpine will increase them.

*Atropine* paralyzes the motor endings of the vagus. An injection of 0.001 Gm. (gr. 1/60) of the latter drug will manifest its action within thirty minutes and disappears in from one to three hours. During the full physiologic action of the drug, it annihilates all reflexes from the stomach, heart, and lungs, hence all symptoms associated with augmented irritability of the vagus are temporarily inhibited. An injection of *pilocarpine* (gr. 1/10) will on the contrary accentuate the sensory and motor neuroses of the vagus.

One may physiologically block a host of reflex cardiac anomalies by an adequate dose of atropine. Thus, a case of angina pectoris vasomotoria may be cited with the following symptoms: heart-signs, chest-pressure, and fear ensuing from exposure to cold. Here, the peripheral vasoconstriction due to cold by increasing blood-pressure stimulates the depressor nerve, which in turn by acting on the vagus causes cardiac signs. By paralyzing this physiologic chain with atropine, the hands may be dipped into ice-water without subsequent symptoms, but the latter reappear after the effects have evanesced.<sup>2</sup>

My investigation shows that *anæsthesia* of a *peripheral area diminishes vagus tone*.

Fliess diminishes dysmenorrhœal pains, Koblauck, labor pains, and Siegmund, gastric pains, by nasal cocainization and thus, as they suppose, establish an association between the nose and the stomach and uterus. I have found it impossible to excite the stomach or intestine reflexes, when any mucous area is cocainized (nose, urethra, rectum or mouth).

My clinico-physiologic investigations show further that *visceral*

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<sup>2</sup> *Journal A.M.A.*, March 11, 1911.

<sup>3</sup> *Wiener klin. Woch.*, xxiii, 274, 1910.

*tone is the resultant of not one, but of a summation of peripheral sensory stimuli, and that the continuity of tone may be blocked by annihilation of a single stimulus.* We have shown that pilocarpine increases vagus tone, that atropine annihilates it, and that adrenalin, by stimulating the sympathetic fibres, puts the latter into a state of increased tonus, thereby resulting in a relative reduction of vagus tonus. Augmentation and reduction of vagus tonus may be obtained in a simplified and more expeditious manner by paravertebral pressure, employing an instrument which I have called radicularpressor. The two prongs of the instrument are separated by a distance of 5 Cm. It is designed for diagnostic and therapeutic purposes and with it one may make bilateral pressure on the roots of the spinal nerves at their exit from the intervertebral foramina. The points of exit of the spinal nerves are relatively superficial. Thus in a number of measurements, the exit at a point corresponding to the seventh cervical vertebra was at an approximate depth of 2.6 Cm.

If one makes pressure (the prongs approximating the intervertebral foramina on both sides) at a point corresponding to the seventh cervical spine, vagus tone is increased, and decreased or abolished when pressure is applied at a point between the third and fourth dorsal spines. Pressure is maintained for about a minute.

It is assumed that at the former point the pressor, and at the latter situation the depressor fibres of the vagus are stimulated. During the time pressure is made at the seventh cervical spine with the radicularpressor, a number of vagal phenomena caused by stimulation are observed. I must refer to my book<sup>4</sup> for a detailed description of the latter. To one only will reference be made and that is, a descent of the lower lung border (lung reflex of dilatation). The lower border is first determined by percussion, after which pressure is made for one-half minute and the border again determined. In the norm the descent is about 4 Cm. In vagus hypertonia, it may descend 6 Cm., and in hypotonia, it may descend only 2 Cm., or not at all. Pressure between the spines of the third and fourth dorsal vertebræ causes the lower lung border to recede. Increased vagus tonus is generally associated with a low lung border and its converse condition with a high border.

The visceral and circulatory phenomena observed after an hypo-

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<sup>4</sup>Spondylotherapy: 3d edition, 1912. Philopalia Press, San Francisco.

dermatic injection of adrenalin may be duplicated by applying the electrodes of a rapid sinusoidal current on both sides of the neck between the second and third cervical spines. This point corresponds to the exit of the *phrenic nerve* and I assume that the latter, through its phrenico-abdominal branches which supply the adrenal glands, augments the adrenal secretion.

Among the diseases caused by vagus hypotonia may be mentioned: diabetes, hyperthyroidism, aneurism, certain form of emphysema, etc. Among hypertonic diseases may be mentioned: hypothyroidism, asthma, gastric and intestinal neuroses.

I shall endeavor in subsequent lectures to show you how many diseases rapidly respond to treatment based on the assumption that they are caused by either an increase or a decrease in the tone of the vagus. In concluding this lecture permit me to recapitulate the essential facts which we have discussed.

In the norm the vagal and sympathetic fibres are in physiologic antagonism. Vagal stimulation is achieved by the use of pilocarpine or by paravertebral pressure corresponding to the seventh cervical spine.

Sympathetic stimulation is effected by the hypodermatic employment of adrenalin and sinusoidalization of the phrenic nerve.

Vagus tone is diminished by sympathetic stimulation, by atropine, and by the barotherapeutic manœuvre of stimulation of the depressor nerve. Symptoms or diseases (asthma, angina pectoris) due to increased vagus tone are accentuated by the pharmacologic and mechanic methods which increase vagus tone, and by inhibiting the latter the symptoms are ameliorated.

Many drugs owe their efficiency, as we have shown, by either increasing or decreasing the tone of the vagus. The scientific study of pharmacology should not be limited to laboratory animals; on the contrary, the human offers a fruitful field for research work along new and original lines of what I have neologized as *clinical pharmacology*.

My next lecture will be devoted to one of the most typical diseases caused by vagus hypotonia, *viz.*, exophthalmic goitre, and I shall endeavor to show you how, by the employment of methods which increase vagus tone, the treatment of this disease has become simplified and practically specific in its results.

## THE MODERN TREATMENT OF GOUT <sup>1</sup>

BY MARCEL LABBÉ, M.D.

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GENTLEMEN: Gout seems to be a malady as old as the world itself, and a panacea for it still remains to be found, but with a better knowledge of its pathogenesis and with the improved therapeutic agencies at our disposal we claim to be able to treat it more successfully than was formerly done.

During an acute attack any of the familiar liniments containing chloroform or similar ingredient may be applied locally, and such analgesic drugs as pyramidon, phenacetin, and, in some cases, even antipyrine may be prescribed; but in the salicylates and in colchicine we find the best remedies, even in the most difficult cases. We usually use a tincture of the seeds of colchicum, the dose being 1 to 2 grammes (15 to 30 grains) diluted with a glass or more of water, two or three times a day. In order to avoid a possible irritation to the stomach or intestines, we often give a hot infusion of tilia leaves or of chamomile flowers. This may be kept up for four or five days, the usual duration of an acute attack. As to the salicylates, those of soda and aspirin are both of them palliatives and are specifics for gout and rheumatism. The dose is from 0.50 centigramme to 4 to 6 grammes a day (well diluted) for the soda, but we never give more than 2 to 2½ grammes of the aspirin. In certain very acute cases, where the suffering is intense, a small hypodermic of morphine may be given; we always, however, do this ourselves, never entrusting it to a nurse, and the patient is not told what it is. As these acute cases are usually accompanied by gastric or gastro-intestinal troubles, a strict diet is of the utmost importance. We, therefore, prescribe the chamomile infusion, and milk, to the absolute exclusion of eggs and all kinds of meats, no matter how good the appetite may be.

Gout, in its chronic form, as we all know, is due to the retention

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<sup>1</sup> Clinic reported for INTERNATIONAL CLINICS by Dr. Thos. Linn, of Nice, France.



of purin bodies and uric acid in the system, and in combating this condition we must seek to transform the purin into urea, or at least into non-irritating products, and to dissolve the uric acid and effect its elimination by way of the kidneys and the skin. Numerous agents are continually being brought forward claiming to be the best solvents for uric acid, and great difference of opinion exists among chemists as to the relative value of one or the other of these, some holding to nucleinic acid and others favoring thymic acid, etc.

Two varieties of uric acid present themselves for our consideration: endogenic, due to nutritive phenomena, and exogenic, due to causes which we ourselves bring about, such as high living, and which may be avoided by proper diet.

Oxidation of endogenic uric acid may best be induced by any form of muscular exercise which increases organic combustion, such as running, walking, cycling, rowing, fencing, tennis, etc. Automobiling, however, should be avoided, the constant sitting posture tending to produce fat. Hydrotherapy in all its forms is highly to be recommended; as to massage, we do not much favor it, as it usually benefits the gouty masseur more than his subject.

All sorts of drugs have for a long time been used to produce combustion of uric acid and purin bodies in the system, such as benzoate and carbonate of lithium; alkalines, sodium phosphate, and bicarbonate; and, more recently, solurool, piperazine, urotropine, urodonal, and, finally, atophan, have been added to the list. We find benzoate of lithium, in doses of 0.50 centigramme to 1 gramme a day, with the addition of plenty of water, very useful. Luff, in England, and Fauvel, in Paris, claim that salicylate of soda is the best agent for reducing excess of uric acid and purin in the system, but, as their experiments were made on healthy subjects, we doubt whether gouty ones would respond so well. The latest tests seem to indicate that this drug has a decided effect upon the liver, and to this its beneficial action may be due.

Chemical analysis and clinical experiments conducted in this hospital have shown that the latest drug, atophan, stimulated the elimination of purins from 0.67 to 0.86 and that solurool gave 49 to 86 centigrammes of purins; the best results, however, were obtained from aspirin, which, given in daily doses of 2 grammes, for six days, eliminated 49 to 97. Notwithstanding the objections to their use,



the salicylates in general should be retained as the best agents we have for combating gouty diathesis. By prescribing the plentiful drinking of water we can bring about a satisfactory elimination and solution of uric acid, and in our wards we have had occasion to observe the effects of this on a very gouty, obese patient. Given one quart of water a day he passed 40 centigrammes of purin, and by taking two quarts daily he passed 68, and with three quarts 89 centigrammes, so that by doubling the quantity of water we doubled the elimination, and this without any change in diet! So make your gouty patients drink plenty of water. The simple natural mineral waters of Evian and the more diuretic waters of Vittel and Contréxville are to be recommended. In the hospital we use large doses of hot infusions of tilia leaves, chamomile flowers, cherry stems, broom, etc.

Some writers have lately called attention to the use of inhalations and intravenous injections of the radio-active emanations of certain mineral waters in the treatment of gout. A current of water is passed through water highly charged with radio-active substances, and these are carried into the inhalation rooms. The oxygen permits of good breathing and prevents danger of asphyxia. Thus far the results have been an aggravation of the trouble followed by marked improvement, as is usually the case when the cure is taken at the springs themselves, when the acute symptoms brought on by the treatment are succeeded by improvement, the old idea of driving out a malady.

We do not wish to pronounce judgment on this new treatment, but mention it merely to acquaint you with the latest method in the treatment of gout. We have seen cases where patients under the influence of these emanations have been relieved of their excess of uric acid, and we ourselves are experimenting with nasal inhalations of air that has been passed through a solution of radium bromide.

The modern treatment may be formulated as follows: During ten days in each month we give 2 to 3 grammes of a salicylate a day. For the next ten days we prescribe one quart of Vittel or Contréxville water, preferably hot, every half hour or hour, a glass or as much more as can be taken at a time, and, if possible, to be taken in bed in the morning, the recumbent position being best for the treatment. At night we give about one pint of a hot infusion of cherry stems or broom.

The diet should avoid all foods rich in purins, such as meat, game, sweetbread, liver, kidney, brain, and the internal organs in general. Vegetables, with the exception of leguminous ones such as peas, beans, etc., and spinach, on account of its constituent oxalic acid, may be given. Tea, coffee, and chocolate, being rich in uric acid products, are forbidden. On the whole, the diet should be a lacto-ovo-vegetarian one, with the exceptions as already indicated. Alcoholic drinks should, as a rule, be avoided. It is a well-known fact that one glass of champagne or red wine is enough to bring on an attack of gout. A little white wine, diluted with water or Moselle, may, however, be permitted. Beer is very harmful. Cider, on the other hand, may be recommended in many cases. Immediately after an attack the diet should be almost exclusively a milk diet, with an occasional egg and some fresh cheese, such as cottage cheese.

In cases of arthritis, where the swellings are not painful and where acute attacks are rare, if the patient complains of weakness, we allow a small quantity of meat once a day or once every other day, with the noonday meal. Sweetbreads and liver should be studiously avoided, as they are actual poison for the gouty.

If your patients will rigidly and persistently follow the cure as outlined above, paying particular attention to drinking large quantities of water, and keeping the diet strictly, they will, without a doubt, be much benefited, for it is only by a hygienic mode of life that we can secure good results, since a specific cure for gout is as yet unknown.

# THE TREATMENT OF CHRONIC ENDOCARDITIS

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It is difficult to estimate the exact increase in the number of people affected with diseases of the circulatory system.

Tables of vital statistics in the registration areas show a steady diminution in the proportionate number of deaths from the various communicable diseases, due, no doubt, to better methods of sanitary control. On the other hand, there is apparently an enormous increase in the proportionate number of deaths from diseases of the heart, arteries, and kidneys. Perhaps a large amount of this increase is due to a different classification of the causes of death, to better methods of diagnosis, and to more accurate filling out of death certificates.

The total deaths from pulmonary tuberculosis in New York State in 1888 were 12,390, and in 1908 had only increased to 14,347, and the ratio per 100,000 living had decreased from 208 to 167.

On the other hand, the deaths from diseases of the circulatory system (including heart-disease and diseases of the arteries, and not including nephritis) increased from 6394 in 1888 to 17,833 in 1908, and the ratio per 100,000 living increased from 105.8 to 216.1.

## NEW YORK STATE STATISTICS.

Total population .....	5,946,248	7,958,459	8,546,356
Total deaths .....	114,584	122,584	138,912
Deaths pulmonary tuberculosis .....	12,390	12,979	14,347
Deaths pulmonary tuberculosis per 100,- 000 living .....	208.3	183.8	167.5
Per cent. of deaths tuberculosis to total mortality .....	10.8	10.7	10.3
Deaths diseases of the circulatory system	6,394	10,511	17,233
Deaths diseases circulatory system per 100,000 living .....	105.8	148.5	218.1
Per cent. of deaths diseases circulatory system to total mortality.....	5.5	8.5	12.4

Deaths genito-urinary diseases .....	4,926	8,641	11,329
Deaths genito-urinary diseases per 100,- 000 living .....	82.8	122.4	132.5
Per cent. genito-urinary diseases to total mortality .....	4.2	7.04	8.1

It would seem, therefore, from these figures that there was a real increase in these diseases. This is notably true of heart-disease of the death record, or the chronic endocarditis of the clinician.

With an increased mortality from chronic endocarditis, it is necessary to be more watchful in prophylaxis, more skilful in diagnosis, and more careful in the treatment of this distressing disorder.

PROPHYLAXIS.—With such a progressive and incurable disease, prophylaxis would not seem to offer promising results. Such, however, is not the case. Much can be done to prevent endocarditis by the proper treatment of acute rheumatism. So many cases of acute rheumatism are neglected because it is difficult for doctor or patient to be impressed with the future danger to the heart-valve or muscle by a few fleeting joint pains and an occasional rise of temperature. In instances of this kind the patient is allowed to go about—and at evident risk to his valves or myocardium.

During the winter and spring there are many such cases among the poor of our cities, who only apply for dispensary relief, and are either not considered ill enough for hospital care or are refused admission to the hospitals because of their overcrowding with graver and more acute ailments. These patients, no matter how much they may object, should be kept at rest and urgent antirheumatic treatment administered until pain, fever, and all other symptoms have disappeared. During convalescence from acute rheumatism, extreme watchfulness should be given to the heart, whether it has been the site of previous disease or is apparently normal. If no evident murmur is present and if there are no evident signs of cardiac insufficiency, the physician too often curtails convalescence and allows too much exercise, forgetting that severe acute rheumatism hardly ever leaves the myocardium unharmed.

That the myocardium must be considered, whether or not a valvular defect is present, has been shown by many authors. Krehl<sup>1</sup> and Romberg<sup>2</sup> pointed out that chronic inflammatory changes were usually present in the heart-muscle. Poynton,<sup>3</sup> Paine, Coombs,<sup>4</sup> and

others of the English school, have drawn attention to the frequent presence of rheumatic nodules in insufficient hearts, and, more recently, Aschoff <sup>5</sup> and Tawara <sup>5</sup> and others have shown how frequently the paths of conductivity are damaged.

A few weeks' longer convalescence, the gradual return to active exercise and work, whether there is evidence of valvular disease or not, may put off for years the ever-threatening cardiac insufficiency.

The more careful management of the milder forms of acute rheumatism \* is urged and a longer and more careful convalescence of the severer cases.

Every patient with an acute tonsillitis, especially if the patient be a child, should have a careful cardiac examination made, for not infrequently an unsuspected cardiac lesion will present itself, and the proper advice may be of much benefit in preventing cardiac insufficiency.

Care should be taken not to mistake a systolic apical murmur, so frequently heard in fever, for the murmur of a true endocarditis.

Not only in aortic aneurism, but also in those cases of aortic valvular disease so frequently occurring in men toward the middle term of life, syphilis should always be suspected. Neglected and untreated syphilis is a frequent cause of endocarditis, and the thorough treatment of syphilis will be of value in preventing disease of the aortic valves or the aorta, with the usual subsequent dilatation of the aortic ring. With the onset of aortic valvular disease of non-rheumatic origin, a Wassermann test should be made and, if positive, the patient should be given prompt antisyphilitic treatment.

*General Hygiene.*—It is easy to advise simple nourishing food, good habits, avoidance of excessive hard work, abstinence from or moderation with alcohol and tobacco, freedom from worry, and so on; but, with our modern complicated mode of life, it is often difficult to carry out this advice, and the physician's first opportunity to give such counsel is often years after the damage has been done. Nevertheless, it is the physician's duty to counsel wholesome methods of living when opportunity offers, especially to those patients who have

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\* I purposely avoid the terms "acute articular rheumatism" and "acute rheumatic fever," for rheumatism and its complicating endocarditis may occur without fever.



already incurred risk from an attack of rheumatism, tonsillitis, or syphilis.

In the after-care of the acute infectious diseases it is of extreme importance to maintain a watchful eye, especially over patients convalescing from diphtheria, scarlet fever, typhoid, and chorea. In all of these convalescent conditions the physician and patient must coöperate with each other, and the physician must explain the reason for prolonging the convalescence and after-care.

The patient's confidence and coöperation must be obtained, and much may be gained by informing him in simple language the nature of his condition. It is the patient's right to know what his condition is, and all directions may be of no avail unless he knows the nature of his trouble. He need not be told roughly, "You have heart trouble and you had better take care of yourself," but he may be told gently that "some time ago you had an illness which damaged your heart-muscle or one of your heart-valves, and that has somewhat weakened or has somewhat impaired your heart's action." Then the patient may be told that his heart is or is not acting as it should, and that, if such and such precautions are taken, he may never have any serious trouble.

No harm will result from informing the patient that he has heart trouble, and in neurasthenics their confidence will be gained by being honest. Both the patient's and the doctor's interest demands it.

**TREATMENT OF ENDOCARDITIS ITSELF.**—Accurate diagnosis is of the greatest importance, and the diagnosis must be not only anatomical, but functional and etiological. It is useless to believe that thickened, sclerosed, calcified, ulcerated, or perforated valves can be restored to their original condition, and all treatment with this end in view will be of no avail.

There are numerous conditions found at autopsy where the valves are found to be thickened and sclerosed and where there evidently has been a true endocarditis, but where the valves have remained sufficient. In such cases as this there may be no associated hypertrophy, and such a condition may never be diagnosed during life. Such a condition would not, of course, call for treatment. Where the muscle is hypertrophied and perfect compensation exists, the balance of reserve force may be increased if the muscle has not been damaged; and some patients with chronic valvular disease may go



through life without the slightest cardiac insufficiency. In others the balance of reserve force may be the same as that of the normal heart, but in by far the larger majority of cases the reserve force is diminished. If this force is diminished it means that the patient cannot go to the same limits of physical exercise to which he was able before his heart became damaged.

The treatment, therefore, resolves itself into the treatment of the endocarditis and the associated heart-muscle by measures looking toward the maintenance of a sufficient balance of reserve force on the part of the heart-muscle. Romberg<sup>6</sup> and Krehl<sup>7</sup> have stated that in hypertrophied hearts the balance of reserve force was increased. This is true theoretically and true in a limited number of cases where no associated muscular lesion exists. In considering the maintenance of this reserve force in hypertrophied hearts, one must always remember that clinically the muscle of the hypertrophied heart associated with some valvular lesion is practically never normal.

The treatment of endocarditis proper, excluding the specific treatment previously mentioned, is without value, except in efforts made to prevent further damage to the valves, but much may be done by prompt and efficient management of intercurrent infections, and these may be prevented oftentimes by extra attention to personal hygiene and by the maintenance of the reserve force of the heart-muscle.

*Can This Reserve Force be Maintained or Increased?*—The reserve force can best be maintained by carrying out the general hygienic and prophylactic measures just described, and by care taken to prevent overstrain of the heart-muscle. If the reserve force is good, the heart, like other muscles of the body, will remain most efficient when properly exercised, and many patients with serious valvular defects will remain in better condition if they are allowed to pursue the open-air sports to which they have been accustomed, provided always that they stay within the limits of fatigue and provided that they do not suffer from cardiac symptoms. Many patients, especially those of the more well-to-do classes, if their reserve force be good, will do better if allowed to enjoy walking, fishing, shooting, riding, and other outdoor sports.

In a large number of cases the reserve force can be increased, and chiefly by the proper exercise of the heart-muscle. Work for the heart-muscle is brought about by physical exercise, and the most

natural exercise is walking. Walking is often given the dignified term of "graduated exercises," and the direction of the walks or "exercises" is often left to the physician at a foreign or native health resort. One may walk definite distances on a country road or in a city square under the direction of the physician just as well as in some agreeable health resort. The following case is an example:

H. C., male, twenty-seven years of age, has had rheumatism, scarlet fever, and chorea. Dyspnœa, œdema, and cough, in February, 1908. He had, on examination, signs of mitral and aortic valvular disease. After rest and digitalis, much improved, returned to work as porter in April, 1908. In June, 1909, gave up work on account of dyspnœa and weakness. During July, August, and September was up and about, dyspnœa on exertion. September 23d patient was in bad condition, auricular fibrillation, dyspnœa, cyanosis, pulsating liver, etc. Was given full doses of digitalis, which, after marked improvement, was stopped October 5th. On October 19th he was put on graduated exercises, first to walk half way down the block from his home and return. Walks were gradually increased and stairs were added, and after six weeks' time he was able to walk half a mile daily without dyspnœa. By February, 1910, he was able to walk a mile a day and go up two flights of stairs without dyspnœa. By May, 1910, walked two miles readily without dyspnœa, and this result without medication.

November, 1910, although he has been advised not to work, he can walk about practically all day and go upstairs without being tired or dyspnœic.

October, 1911, patient is able to be about all day, has spent summer in sailing, motoring, and so on.

Here was a patient, then, who in 1909 had to give up work and, from then until the following September, had constant dyspnœa on exertion. By means of graduated exercises in his own city he has been able to progress so that he is now able to be up and about all day and to do practically all the walking he desires; and, although his compensation is not perfect and probably never will be, he keeps in good health and is comfortable. During this time there was never any appreciable change in the size of the heart by ordinary methods of examination. The marked arrhythmia persists.

Results similar to this are frequent and are often seen among the less wealthy people, who are unable to go away to take a "cure."

If exercise is of value it must not be understood that rest is not also of value. In the beginning the exercise must be kept well within the limits of fatigue and the patient must be kept under constant supervision; the rate of pulse, the amount of dyspnœa, and the daily

weight for the first few weeks are of great importance. The pulse should not rise to more than twenty beats above normal during exercise, and should return to normal soon after. If the increased rate persists after fifteen minutes' rest, or if there is dyspnœa or cardiac palpitation, or pain, too much has been done, and when walking is again attempted it should be of shorter duration. Further, these walks should be begun on the level and not against a high wind, nor on excessively cold days. As progress is made, the patient should be cautioned not to make too sudden or prolonged efforts. In a few minutes of severe exercise, all that has been gained after months of effort may be lost, and each time that ground is lost it is more difficult to be regained.

In some cases where the least effort in the way of walking or physical exercise gives rise to dyspnœa or palpitation exercise may be given by means of massage. General body massage, beginning at twenty minutes and increasing to half an hour or an hour, should be given for a week, and if this is well borne, then resistance exercises may be given by a skilled masseur.

*Indications for Massage.*—After convalescence from a cardiac insufficiency, when what is commonly called ward, chair, or rest compensation exists, days and weeks may elapse without any improvement. The patient feels too well to stay in bed, and after being up all day is a little tired at night, the heart-rate is a little more rapid, and there is some œdema, shown by a gain in weight or by some puffiness of the ankles. In these cases the passive massage movements are often of value in improving the heart's force.

If any improvement has resulted from massage, further improvement may sometimes be obtained by passive movements of the arms and legs. These passive movements apparently do not cause any acceleration of the pulse, and after a week's time resistance movements may be substituted. Resistance movements may be used, and sometimes to advantage, in association with massage, and passive movements before the patient is able to walk, or when he is able to walk but little. These methods of treatment are chiefly used at the various cures on the Continent and are generally known as the Schott<sup>8</sup> treatment, which was for many years carried out by him in connection with the carbonic acid baths at Nauheim.<sup>9</sup>

*Exercise.*—As has already been mentioned, it is wise to allow

patients to indulge in outdoor sports, provided the strength of their heart-muscle permits it. Oertel<sup>10</sup> suggested gradual hill-climbing to increase the strength of the heart, but unless carried out under careful supervision at a cure it is wiser not to allow patients to attempt hill-climbing, because they are too easily tempted to overdo it, with serious results. Young people will often want to indulge in dancing, and this may be allowed in moderation, but here again there is a temptation to overdo it. The more severe exercises, such as running of races, football, and tennis, should be interdicted, although there have been students at some of the smaller colleges, where physical examination has not been compulsory, who have played on the football teams for one or more seasons before it was learned that a serious valvular defect was present.

*Work.*—In some instances it may be necessary for the patient to change his occupation or mode of life in order to prevent unusual cardiac strain. The work of blacksmiths, iron-workers, longshoremen, and so on, is so severe that the heart may be seriously overtaxed.

In our large cities many patients live in tall buildings where there are a number of stairs to climb each day. This stair-climbing may or may not be harmful, and if harmful the patient should be instructed to live on the ground floor. With the wealthier class, especially in elderly people, where cardiac insufficiency always threatens, a total abstinence of exercise by use of elevator and automobile often prolongs the onset of a cardiac insufficiency.

*Treatment by Baths and Cures.*—The question of a cure is continually coming up, and many patients are continually going to various resorts, some with marked benefit and some without any. For a limited number of cardiac cases cures are of value, but they must be for selected cases, and the following questions must be answered before a patient should be sent to a cure:

Can this patient be further benefited at home?

Will he be benefited by a vacation at the seashore, mountains, or country?

Can he afford to undertake the expense of the journey and the cure?

There are many individuals who, in the summer time, would refuse to take more than two or three weeks' vacation, yet who would be willing to go away for two months for the purpose of taking a



cure. Men who are in active life and who insist upon keeping in touch with their affairs are often much benefited by the complete change which a cure gives them; and those who are utterly bored at the idea of doing nothing and who—when on a vacation—overeat, overdrink, and overexercise, are much better off if they spend their vacation time at a cure. It is difficult to lay down hard and fast rules in the selection of patients. First of all, they must be well-to-do, and they must be sufficiently strong to take the journey. Almost every heart where compensation is not perfect, and yet where there remains a considerable balance of reserve force, will usually be benefited by a cure. The patient must, however, belong to that class who can leave home without worry and without undue expense, for the advantages of the cure are not so great but that a cheaper substitute could not be given at home.

*Selection of the Cure.*—Most important of all is the selection of the patient's physician at a cure, for without good medical care and supervision all the advantages of such a treatment may be lost. Second, what cure should be selected? For business men of large affairs the cures of this country are of little value, for there are too many temptations to keep them in daily touch with their affairs. Telephones, telegrams, letters, and newspapers do not allow the mental rest that should be obtained at a cure. One should also have in mind, in selecting a cure, the question of suitable accommodations and the possibility of meeting people of one's own walk of life. The advantages of the cure consist in freedom from business and household cares, body and mental rest, ease in obtaining a suitable diet, the daily routine, pleasant surroundings, walks, drives, and music. The most famous of all the cures for heart trouble is at Bad Nauheim, in Germany. There the physicians believe that marked benefit is obtained by warm carbonic acid baths, massage, resistance exercises, and graduated walks. There is a considerable difference of opinion between medical teachers as to whether or not there is any actual advantage in the baths.

Huchard<sup>11</sup> believes that many physicians at these health resorts deceive themselves by incorrect methods of diagnosis and claim cures when they consist only in some slight change for the better in the physical condition of the heart, which might occur without any treatment.

Mackenzie<sup>12</sup> believes that tap water at a temperature of 89° F. will produce the same slowing of the pulse that the strong Sprudel baths at Nauheim will. He says, "If the individual is well-to-do and there is not much the matter with him—well, Nauheim is as good a place to send him to as any other."

Krehl<sup>13</sup> says that he has no personal experience with the Schott treatment, but he sees no reason not to believe in it, and says that he has seen a heart diminished in size by resistance movements.

Romberg<sup>14</sup> states that the CO<sub>2</sub> baths increase the capacity of the heart for work. He also states that "the baths are contra-indicated in severe insufficiencies when dyspnœa is present while at rest, and there is general weakness, or when serous effusions are present. The baths also should not be given in severe angina pectoris, nor too soon after compensation has been restored, nor after an acute rheumatism, nor after a fresh bronchitis. They should not be given in aneurisms, nor when there is thrombosis of any vein, nor when there is possibility of cerebral hemorrhage. Care should be taken in the presence of arteriosclerosis, and they are also contra-indicated in cardiac insufficiency from nephritis and in nervous or easily excitable individuals."

The after-cure is also of importance, where the patients enjoy a further vacation before they return to their homes. The most important question is, have they benefited by their treatment and is this benefit lasting?

Many patients feel better at the time, but relapse to their original condition on their return to their own homes. A number of patients have undoubtedly, in the past, been positively harmed by such treatment, but many patients have received lasting benefit, yet there is always the possibility that many of these would have been as much benefited by proper care at home.

*Diet.*—It is not possible to prescribe a standard diet for patients with heart trouble. While the patient is in good condition a full generous diet without meat restriction is the best. A number of things must, however, be remembered. The patient should avoid getting fat, and it may be necessary, from time to time, to curtail carbohydrates in the diet. The patient should also be careful not to overload the stomach, and food should be thoroughly masticated with the endeavor to keep the digestion in good order. With a well-acting



heart it is bad practice to use any special dietary cure, because it may endanger the heart. For example, if a low proteid diet is given, patients are apt to make up their caloric value in carbohydrate food and so take on weight. The increase of weight gives the patient more muscular work and taxes the heart, or there may be an actual fatty overgrowth of the heart, which may embarrass the heart's action. Similarly, a diet of milk alone is to be condemned because the milk diet causes an excess of fluid to be taken, here again giving the heart more work to do, and poorly nourishing not only the body but also the heart-muscle.

In obesity the excess of weight does harm in two ways: First, it increases the body work by carrying extra weight, and also makes the heart action more difficult by fatty overgrowth of the heart or by fatty infiltration in the heart-muscle. In obese cases a reduction cure should be undertaken, the importance of which was first pointed out by Oertel,<sup>15</sup> who restricted the amount of carbohydrate food and also restricted the taking of fluids at meals.

*Alcohol.*—Alcohol is harmful in two ways, and may act either by introducing an excess of fluid into the body or by overstimulating the heart. As a general rule, it is harmful for patients with endocarditis. For those who have depended upon it, a glass of beer or of light wine daily will not cause serious harm, but the majority of people who drink regularly find great difficulty in limiting themselves to a definite amount, and for such patients alcohol had best be abstained from entirely.

Tobacco is also bad for patients, but for many it is useful as a laxative, and a cigar after dinner or a cigarette after breakfast is often conducive to a satisfactory movement of the bowels. For patients with compensated hearts, who have always been accustomed to smoking, a small amount of tobacco is not harmful, and they can usually be limited to a cigarette after each meal and one small cigar in the evening. Where there is any tendency to cardiac pain or palpitation, tobacco had best be left alone.

A patient with endocarditis, whether the heart be compensating or not, must abstain from, or limit the use of, alcohol and tobacco.

*The Bowels.*—The bowels should be kept in normal condition by a daily movement, and every effort should be made to avoid the giving of drugs for this purpose. By the use of fruit, exercise, and regular

hours even a constipated habit of long standing may be cured. If constipation is obstinate, the milder vegetable laxatives are the most useful—cascara or rhubarb, in from three- to five-grain doses at night. These will be found more satisfactory than the daily giving of salines. Carlsbad salts and other bitter waters may be used occasionally, but the continued use should be avoided.

*Climate.*—Fresh air, as in all other chronic diseases, is of distinct advantage in maintaining the general health, and should be obtained by night as well as by day.

Climate is of undoubted value for many patients with heart trouble for the compensating case; and by a compensating case is meant one who is fully able to do his work. The climate in which one lives and works should be sufficient. The many cases whose reserve force is poor soon find that they are unable to go to and from their work in the face of high winds, that they cannot walk uphill, and that, in severe cold or damp, foggy weather, they suffer from dyspnoea and palpitation or pain. Many of these individuals in a suitable climate can exercise and walk about without the slightest inconvenience.

The ideal climate for such a patient is one which has an equable temperature, and which allows a large amount of outdoor life. It must be remembered that the advantage of climate must be restricted to the well-to-do, and that not every time a heart-murmur is heard should the patient be sent to a better climate. Many patients who live in our eastern and northern cities will do better to pass a part of the winter, at least, in Georgia or Southern California.

For the wealthy, who can afford to travel, and for whom travel is no unnecessary burden of expense, the shores of the Mediterranean afford many resorts where the climate will be equable. Egypt in January and February is usually pleasant, as are also Algiers and Palermo—although there may be many days of rain while at Algiers—and during March and April Italy and the Riviera are agreeable.

*Altitude.*—High altitude taxes the reserve force of the heart and offers some danger. Numbers of patients with chronic valvular disease go from one end of the United States to the other without suffering the slightest inconvenience or being conscious of any change in their condition.

A patient with chronic valvular disease was cautioned by his physician not to go to any altitude, and was told that anything over five hundred feet would seriously inconvenience him and might cause his death. While travelling abroad it was necessary for him to go over a mountain pass of considerable height, or to make a long detour. He was not told of the altitude of this pass, and was entirely unconscious that he had gone over an altitude of more than three thousand feet.

It is a safer procedure, however, not to allow patients who live in low altitudes to make prolonged stays at an altitude of over one thousand feet.

*Treatment with Drugs.*—In perfectly compensating hearts it must be obvious to all that the drug-treatment directed toward the heart is far worse than no treatment. Delafield<sup>16</sup> states, "If the heart is acting well, treatment makes it worse." Tonics, however, are of value in building up the general body tone.

There are a number of border-line cases, however, where the patient's compensation is good for the normal demands of life, but where there is frequently a little dyspnoea and more fatigue than is normal. In such individuals an occasional course of digitalis, a drachm of the tincture in divided doses for each twenty-four hours for four or five days, or a longer course of 10 or 15 minims of the tincture three times a day for several weeks, every two or three months, will often be found to be beneficial, and the patient will have a better sense of well-being and be able to do his or her work with better satisfaction.

There are also cases which recover promptly during an attack of cardiac insufficiency with judicious management and the proper use of digitalis, only to become again dyspnoeic and oedematous upon any attempt at exertion. Many of these cases receive much benefit from the persistent use of digitalis. Naunyn<sup>17</sup> reported favorable results from the administration of the infusion equal in amount to gramme 0.5 to 0.8 of the powder, in forty-eight hours, with a three- to four-day pause, renewing the digitalis again for forty-eight hours, again a pause, and then, after another course and pause, gramme 0.25 a day alternate weeks.

Kussmaul<sup>18</sup> reported the case of a patient with valvular disease

and recurring attacks of cardiac insufficiency who was able to keep up and about by the continued use of small doses of digitalis. The patient was able to increase or diminish the dose for himself, being guided by an increase in weight or the onset of dyspnœa. The patient took digitalis over a period of seven years, from fifteen to fifty-seven grammes a year—a total of three hundred and five and twenty-nine hundredths grammes—that is, over a period of about 2400 days the patient took, on an average, about two grains a day.

These observations have also been confirmed by Sahli<sup>19</sup> and other German writers.

Schmoll<sup>20</sup> more recently calls attention to the need, in many cases, of the long-continued use of digitalis. He found that doses

CHART I.

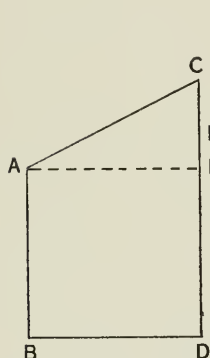


FIG. 1

Normal heart. A, B, Rest force. C, E, Reserve force.

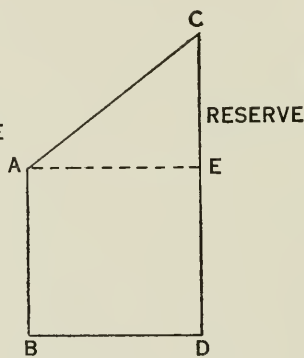


FIG. 2

Athlete's heart, or trained heart. A, B, Rest heart. C, E, Reserve force.

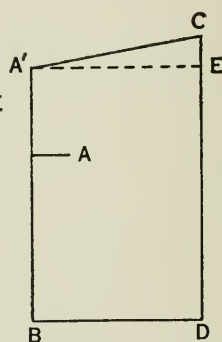


FIG. 3

Cardiac insufficiency. A', B, Rest force. C, E, Reserve force.

of about gramme 0.1 can be taken daily without any bad effect, and that when necessary larger doses of gramme 0.15 to 0.2 can be taken daily over weeks at a time with no unpleasant results.

This tonic use of digitalis must of necessity be limited to those cases of cardiac insufficiency who cannot get along without it, and the longer the use of digitalis can be put off without risk to the patient the better. The unnecessary use of digitalis is harmful, and its use must be reserved to those cases of acute and chronic cardiac insufficiency.

THE TREATMENT OF CARDIAC INSUFFICIENCY.—Cardiac insufficiency means a loss of a part of the normal balance of the reserve

force of the heart-muscle. This may be of mild or severe degree. In the accompanying diagram (Fig. 1) *A B* represents the normal amount of heart-work while the body is at rest and *C E* represents the reserve force of the heart. In the heart of an athlete in training the work of the heart at rest is the same, but the reserve force is increased, as shown by the line *C E*, Fig. 2. When the heart works at a mechanical disadvantage by reason of a valvular lesion, the work required of the heart while the body is at rest is increased, and the amount of work performed by the heart while at rest may be more than double the amount of work performed by the normal heart, and the amount of reserve force in the diseased heart depends upon the gravity of the lesion, as shown in Fig. 3.

A valvular lesion may be present, as evidenced by a loud murmur, but the mechanical disadvantage to the heart's action may be so slight that there is but little diminution in the reserve force. In such an instance cardiac insufficiency may or may not exist, and upon the degree of cardiac insufficiency present depends the amount of treatment needed.

There are many patients with chronic endocarditis who have a variable amount of cardiac insufficiency, who are engaged in various occupations, some of which may cause undue strain upon the heart. This overstrain may bring on an acute insufficiency or may cause the gradual reduction of the reserve force. In these cases it would be of extreme value if it were possible to tell the amount of the reserve force. This as yet seems impossible, though many attempts have been made to determine it. Frequently more can be ascertained from answers to questions like the following: Can the patient go upstairs without dyspnœa? Has he palpitation or pain when walking against the wind? Can he sleep flat on his back at night?—than by listening to the heart's action or to the heart's murmurs.

By means of the changes in the rate and rhythm of the pulse and in blood-pressure after muscular activity various observers have attempted to measure the amount of reserve force by comparing the work done by normal and diseased hearts. Oertel<sup>21</sup> and Rieder<sup>22</sup> found that in normal hearts muscular activity increased the blood-pressure, but that in diseased hearts the blood-pressure either did not rise or fall, and Hüsler<sup>23</sup> found that the heart-rate in diseased hearts was more easily influenced by stress. Gärtner,<sup>24</sup> Jaquet,<sup>25</sup> Speng-



ler,<sup>26</sup> Christ,<sup>27</sup> and Minassian<sup>28</sup> all attempted in various ways to measure the reserve force, and, later, Gräupner<sup>29</sup> and Cabot and Bruce<sup>30</sup> tried similar means, but, although the work of these observers has been carefully done and is of value, yet when tried in individual cases the results of their tests are not sufficiently uniform to be relied upon.

The severe forms of cardiac insufficiency exist when the "work force" has been exhausted or when subjective or objective symptoms of cardiac insufficiency are present when the patient is at rest.

The treatment of acute cardiac insufficiency or break in compensation, or decompensation, should consist in efforts made toward restoring this needful balance of reserve force, and in making these efforts the normal physiology and anatomy of the heart should be kept in mind.

The heart should be relieved of all unnecessary work, and this can be accomplished only by absolute body rest. The patient should be put to bed, in a quiet room, and should be relieved from mental worry, and should not be bothered with outside affairs. Mental rest and absence of emotion are sometimes of as great importance as body rest.

Alcohol should be interdicted and also tobacco.

The diet is described under œdema, which will usually be present in cardiac insufficiency.

*Posture.*—In certain cases of orthopnea the patient is frequently far more comfortable sitting up in a chair, which allows more freedom of play for the respiratory muscles and also allows for a certain amount of blood to be collected in the veins of the legs, which may relieve in part the pulmonary congestion. Many patients, while in bed, sit up, of their own accord, on the edge of the bed with their feet hanging over, and frequently get relief while in this position. Patients often resort to other devices, such as resting their head or hands upon a table or chair.

Patients with chronic endocarditis should be regularly weighed. This will tell not only whether the patient is losing weight from insufficient diet, but also if he is getting too fat from an excess of food. But, more important than this, a gain in weight will herald the onset of œdema before this can be noticed by either patient or physician. A gain in weight of from five to fifteen pounds is pos-



sible without there being any evidence of œdema. But with such a gain in weight there pretty surely will be some other evidence of breaking compensation, and complete rest is at once indicated.

In the treatment of a case of cardiac insufficiency one of the physician's duties is to make himself thoroughly conversant with the position of the heart and to note any change in its position, and also to examine frequently the thorax to determine the presence or absence of congestion or fluid.

If there is any cardiac displacement, or if there is dulness at the right base posteriorly, fluid should always be suspected. When the physical signs are those of atelectasis or hypostatic congestion, remember that it is probably fluid and put in a needle for diagnosis.

*Treatment of Œdema.*—Œdema is treated, first of all, by rest in bed and diminution of the diet. Bowels should be moved daily, preferably by means of a saline cathartic. The best plan of procedure is to weigh the patient daily, to restrict the fluid intake to 40 ounces, and to keep an accurate measurement of the fluid intake and urine output. Normally the urine output will be from five to ten ounces less than the fluid intake for twenty-four hours. If the fluid intake is considerably more than the urine output, it is more than likely that, unless there are frequent fluid movements, water is being retained in the tissues.

An ordinary farinaceous diet is the best for cardiac insufficiency, with restriction of the fluid intake and a restriction of sodium chloride. By this means a sufficiently high caloric value can be given in the diet to insure against loss of protein. Karell<sup>31</sup> suggested years ago that such œdematous patients be given a milk diet, the amount being restricted to one hundred cubic centimetres every two hours, or about one litre of milk in twenty-four hours. After several days Karell gradually increased the amount of milk per day, not allowing any other food, until after six weeks had elapsed. This works well in certain cases; but, while it spares the heart and kidneys, it also causes a loss of body weight, as the caloric value is far too low. The following case illustrates very well the result of Karell's method:

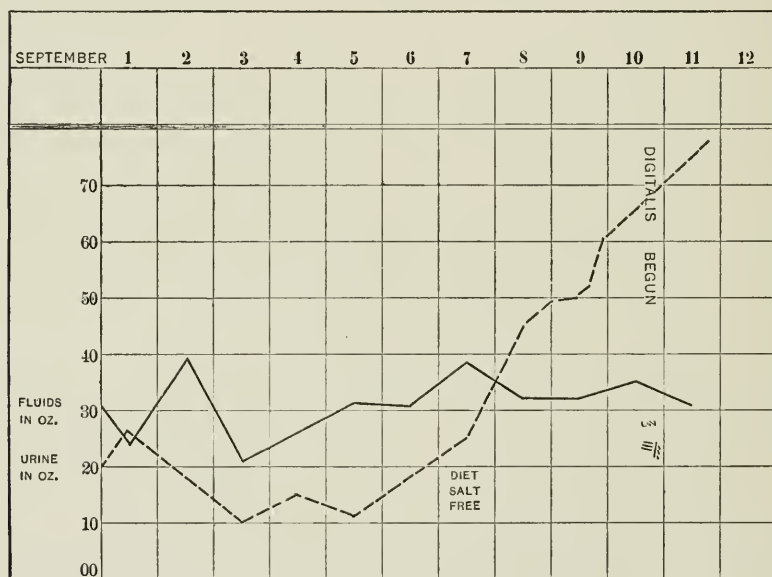
T. B., male, aged 44, colored. Previous history of syphilis, dilated heart, pulsating liver, general anasarca. On admission to the City Hospital, in the service of Dr. Evans, weighed 174½ pounds. Four days later weight was 170, and his total intake of food was then limited to 200 C.c. of milk four times in

the day—at eight, twelve, four, and eight. This treatment was kept up with no medication for seven days. During this time he lost 30½ pounds. He remained in excellent condition, complained of nothing except hunger, and at the end of this time the œdema had entirely disappeared, with the exception of signs of a small amount of fluid at the right base.

Widal <sup>32</sup> suggested the utilization of a salt-free or low salt diet for the treatment of not only nephritic but cardiac œdema. The Karell treatment, of course, gives a limited fluid intake and restricts the amount of salt to less than two grammes. Widal's treatment includes a certain amount of milk and a farinaceous diet of sufficient

CHART II.

DATE \_\_\_\_\_ NAME \_\_\_\_\_ AGE \_\_\_\_\_ PAGE \_\_\_\_\_



Effect of a salt-free diet, with drink restriction.

caloric value, limiting the amount of sodium chloride to two or three grammes per day. It is hard, however, to give a diet which will contain less than five grammes of salt per day.

Magnus-Levy <sup>33</sup> has pointed out that use of the salt-free diet has been made with too little enthusiasm; that it has been tried for a few days and then discarded as worthless. He urges that in cases of œdema a salt-free diet be carried out for weeks, or even months, at times, in order to get a satisfactory effect. The amount of salt

must not exceed three or four grammes a day, for a satisfactory result will not be obtained if an amount as high as five or six grammes per day is given. Better results are obtained, however, in nephritic œdema than in cardiac œdema. But as chronic congestion of the kidneys almost always exists, and as chronic nephritis is often present, it should always be given a trial.

The following case of cardiac œdema is of interest in showing the results of a salt-free diet with drink restriction. The patient had been on large doses of digitalis with gradually increasing œdema. With the onset of gastric symptoms the digitalis was stopped twelve days before the salt-free diet was begun, and without any medication the urine promptly increased, as is shown in the chart. (Chart II.)

The milk-cure is mentioned only to be condemned, except when carried out for a few days at a time, according to the method laid down by Karell. To give a milk-cure for a long period of time without the patient's losing body protein, at least four litres per day must be given to obtain the required caloric value. Four litres of fluid, however, seriously overtax the already overburdened heart and add increased work to the kidneys, presumably already congested.

The restriction of the fluid intake and the measurement of the fluid intake and urine output were first suggested by Oertel, who also restricted the taking of fluid at meals.

Mechanical measures are also necessary for the treatment of hydrothorax and ascites. Physical signs which lead one to believe that hydrothorax is present should be verified by the use of the exploring needle, and when fluid is obtained it should be aspirated, for frequently when the physical signs seem insignificant—slight dulness below the angle of the scapula, diminished breathing, and subcrepitant râles with little or no change in the quality of the breathing or the voice or fremitus—aspiration may withdraw from one to two litres of fluid. For example, a patient was admitted to the City Hospital with advanced aortic valvular disease with marked cardiac insufficiency. The house physician questioned the presence of fluid in the thorax, as the signs at the right base revealed only slight dulness with diminished voice and breathing. A needle was inserted, however, for diagnosis, and, upon fluid being obtained, aspiration was performed and 1620 C.c. of fluid withdrawn, with marked relief of the patient's symptoms.

The withdrawal of the fluid relieves dyspnœa by relieving the mechanical embarrassment of the heart and allowing fuller lung capacity. This measure not infrequently turns the tide in the patient's favor and allows the heart to regain some of its reserve force. Not infrequently fluid reaccumulates without any change in physical signs and without any evident cardiac displacement, but the recurring dyspnœa, with orthopnœa, discomfort, and sleeplessness, will be sufficient indication that another aspiration is needed.

*Ascites.*—Marked enlargement of the liver with œdema of the abdominal wall is sometimes confused with ascites. Ascites is not nearly so frequent as hydrothorax, but when present should be treated on the same lines. Œdema of the extremities is rarely so great as to need any mechanical treatment, although occasionally acupuncture is necessary to draw off the fluid.

*Treatment with Drugs.*—In the treatment of endocarditis where the balance of reserve force is sufficient, drugs directed toward the treatment of the heart do more harm than good. The judicious use of tonics and care of the digestion are, however, of great value. As has been already pointed out in cases where the reserve force is diminished, but no acute cardiac insufficiency exists, an occasional course or tonic doses of digitalis are useful. In acute cardiac insufficiency rest in bed and proper diet may alone be sufficient, but drugs are often, and urgently, needed, and the drug above all which is uniformly of greatest value is digitalis.

*Indications for Digitalis.*—In practically all forms of cardiac insufficiency digitalis is indicated, but results are obtained chiefly in those insufficient hearts where the heart-muscle has been but little diseased. In many instances, however, where the heart-muscle has undergone considerable sclerosis, digitalis is still of great value. In dilated hearts, when a partial or complete block is present, digitalis is at times of no value, and sometimes actually does harm by increasing the block, but is always to be tried if marked insufficiency exists.

Digitalis is also indicated where the rest force is insufficient; that is, when symptoms exist during complete body rest. Theoretically, in aortic insufficiency, it is contraindicated on account of over-diastolic filling. But the condition of the heart weakness exists, and digitalis is of great value and its beneficial effect on the heart-

muscle more than counteracts any theoretical danger of over-diastolic filling. Digitalis is also indicated when œdema is present, and if rest and dietary measures have not reduced the œdema, digitalis is the first diuretic to be used.

The older text-books urge the use of digitalis in rapid and irregular hearts. In many of these cases when digitalis was properly administered it resulted in a marked slowing of the pulse and marked improvement in the heart's rhythm, and in some cases there was no improvement. The work, chiefly of Wenckebach<sup>34</sup> and Mackenzie,<sup>35</sup> has shown that many cases of perpetually irregular and rapid pulse were due to fibrillation of the auricles, and in these cases Mackenzie<sup>36</sup> has shown digitalis to be of great advantage by diminishing the conductivity and causing a partial block.

Since Williams<sup>37</sup> first showed, experimentally, that digitalis increased the blood-pressure in the frog, this observation has been verified time and again, so that the teaching has been that digitalis was harmful in cases of increased blood-pressure and in arteriosclerosis, both on experimental and theoretical grounds. There was universal agreement against the use of digitalis in those cases of failing compensation associated with high blood-pressure. Unfortunately, these observations still exist in most books on pharmacology and therapeutics. Even Cushny,<sup>38</sup> in 1911, writes that "in these cases digitalis is to be used with caution, and perhaps strophanthus is to be preferred to digitalis." He goes on to say, however, that "a high blood-pressure ought not to be regarded as definitely contraindicating the use of digitalis and its allies, however, for excellent results often follow . . ."

Students, however, are still being taught that digitalis must not be used in cases of hypertension, on account of the danger of intracranial hemorrhage.

Sahli<sup>39</sup> was the first to report, in 1901, that, instead of always raising the arterial pressure, digitalis at times actually lowers the pressure 30 to 40 mm. Hg.

Sahli thought that the tension was increased as a result of over-stimulation of the medulla by products in the blood, presumably CO<sub>2</sub> and other substances, and that the administration of digitalis so much relieved the dyspnoea and cyanosis that this stimulation of the medulla was removed, and he suggested, also, that the finer



arterioles and capillaries would be more widely opened as a result of the increased force of the heart's beat, and the blood-pressure was thus reduced.

Janeway <sup>40</sup> quotes the observations of Christeller, Frenkel, Heike, Hensen, Gross, and Potain, and states that "all fail to find any relation between the arterial tension and the circulatory improvement from digitalis. This point is important, for it makes it clear that the effect of many circulatory drugs, of undoubted stimulant properties, will not be visible on the pressure chart; because altered blood-distribution, and not increased blood-pressure, is their best result."

Müller <sup>41</sup> more recently showed that there was little or no action on the blood-vessels, after the injection of digalen or strophanthin when given in doses sufficient to produce a full digitalis action.

Schmoll <sup>42</sup> also writes that: "Many writers advise the giving of vasodilators to counteract the constrictor effect of digitalis. It is high time that we discard this hoary tradition and look squarely at the facts in the case. As mentioned at the beginning of this article, most of the work done on digitalis was with toxic doses, and vasoconstriction is one of the toxic effects. It has been shown clinically that in the majority of patients with beginning digitalis effect the blood-pressure falls."

*Contraindications.*—Digitalis should not be given for tachycardia or for irregularity alone, unless there be signs of cardiac insufficiency. It is also contraindicated in irregularity or slowness due to heart-block, though it may sometimes be tried when the heart-block is associated with marked cardiac insufficiency. It may seem surprising to some to insist that digitalis is contraindicated with rapid and irregular hearts, but it must be insisted upon that digitalis is a drug primarily for cardiac insufficiency and that it is contraindicated in cardiac disease, unless insufficiency be present. It must be remembered that digitalis is *not* contraindicated in the arteriosclerosis or in hypertension when associated with cardiac insufficiency.

*Preparations and Administration.*—The most trying thing in digitalis therapy is the fact that digitalis is so often unreliable, and on account of the unreliability of the ordinary well-tried preparations, the infusion and the powder, numerous others have been made, which, though they in themselves are not always reliable, are often better than one of the older preparations. The best preparations are the



infusion and the tincture. In administering digitalis there can be no hard-and-fast rule, except that the physiological effect must be obtained. The giving of so many minims of the tincture or so many ounces of the infusion, therefore, cannot be stated dogmatically, and if the physiological effect is not obtained by use of the infusion or tincture when given in maximum doses, the only alternative is to seek a more reliable apothecary or to administer one of the substitutes, such as digalen or digitalin.

The most consistent results will be obtained by the administration of the infusion, freshly made from English leaves. It should be given in divided doses, amounting to three ounces a day, for four or five days, or longer, until the desired physiological effect is produced. As digitalis is cumulative, the dose should be diminished as soon as this desired physiological effect ensues; that is, slowing of the heart-rate and diuresis. If the dose is then diminished the unpleasant gastro-intestinal symptoms are often avoided.

If the infusion cannot be obtained fresh, the tincture may be given in doses of one drachm per twenty-four hours in divided doses, for four or five days, and again diminished. These two preparations should be the ones first tried when the drug can be administered by mouth and when there is time to wait for the effect.

The powder is often used, especially in a combination known as the Trinity pill; one grain each of powdered digitalis, powdered squill, and calomel. In many cases of œdema the pill is of value, but, where nephritic complications exist, mercurial preparations should be given only after considering always the possibility of salivation. The pill is given once a day at night.

For more prompt digitalis effect, hypodermatic or intravenous administration is necessary, the tincture may be given in five- to ten-minim doses hypodermatically, but it is extremely irritating. Digalen (Cloetta) may be given either hypodermatically or intravenously in doses of five to ten minims three times a day.

Glucosides of digitalis are sometimes used hypodermatically, but, on account of their unreliability, cannot always be depended upon. The tincture of digitalis is often used hypodermatically, but should only be administered under urgent conditions, as the digitalis is extremely irritating and very apt to cause infections. It is, however, of great value where medication cannot be administered by

the stomach and in extreme cases of cardiac insufficiency where a prompt action is needed.

Other drugs of the digitalis group, which are of use at times, are strophanthus and squills. *Convallaria* and *apocynum* have had some reputation in the past in the treatment of cardiac disorders, but are now infrequently used. The action of strophanthus is very similar to that of digitalis, but less frequently is its action satisfactory. Strophanthus is usually better borne by the stomach and is supposed by some to have less effect upon arterial tension. It is wiser to make the first trial of digitalis, and, in case digitalis fails, strophanthus may be tried. The glucosides of strophanthus have been used, especially strophanthin and ouabain. These are both suggested for hypodermatic and even intravenous use, but are attended with risk when used in the later way.

Fraenkel<sup>43</sup> has injected, intravenously, 0.75 mg. of strophanthin and has noted remarkable results occurring in a very short time. Romberg<sup>44</sup> warns against its use on account of the possibility of sudden death, and states that if it has to be used it should not be used immediately after or during the administration of digitalis, and urges that at least a week should intervene between the time of the last dose of digitalis and the intravenous administration of strophanthin.

In cases of acute heart-failure such stimulants as alcohol, caffeine, and camphor are of great value. If medication can be taken by mouth, a drink of spirits, brandy or whiskey, followed by a cup of clear, hot coffee or tea, has great stimulating value. It will also be of value to give a pint of hot coffee by rectum, with the addition of a certain amount of alcohol in the form of spirits. If a quicker stimulant is needed, a grain of camphor dissolved in ten minims of olive oil may be given hypodermatically or a grain of camphor dissolved in ether; this last is irritating and may cause local death of the tissues.

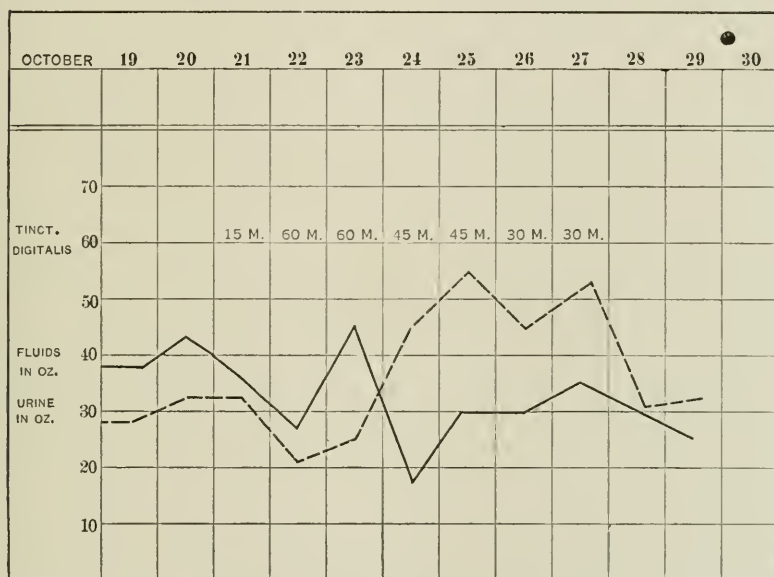
Drugs of the diuretic class will often have to be used for the treatment of œdema. It is urged, however, that in every case of cardiac insufficiency rest, dietetic measures—such as those suggested by Karell, Oertel, and Widal—and the proper use of digitalis should be thoroughly tried before making any attempt to reduce œdema by diuretics. One is too apt to use drugs for general measures and,

especially the inexperienced, to use the many diuretic drugs—often with good results—but it is a much more natural procedure to try other measures before stimulating the kidneys to increased activity. Many cases of cardiac œdema are still being treated with diuretics and at the same time are allowed a liberal diet or a full fluid diet.

The preparations of the caffeine group are of distinct value, especially the sodium salts of caffeine and theobromine and theophyllin. Theocin, an artificial preparation of theophyllin, is also used. All

CHART III.

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Effect of digitalis on the secretion of urine.

of these preparations possess one distinct disadvantage. They are extremely irritating to the stomach, many patients showing a marked idiosyncrasy to them—and they have to be used with care and often withheld. It must not be forgotten that digitalis itself is one of the most satisfactory diuretics of all. The diuretic action of digitalis has always been explained by the fact that the time-volume flow of the blood through the kidneys was increased. The recent work of Hedinger<sup>46</sup> has shown that digitalis has a marked local diuretic action on the kidney itself.

The following case shows the effect of digitalis on the secretion of urine. A patient with advanced cardiac insufficiency began to pass smaller and smaller amounts of urine, and when œdema began to be present, a salt-free diet causing no increase in the amount of urine, he was given the tincture of digitalis four times a day. This was diminished as soon as the diuresis effect was obtained. The accompanying table shows the prompt rise in urinary flow after 135 minims of the tincture had been administered. (Chart III.)

*Treatment of Symptoms.*—Symptoms are the result of cardiac insufficiency, and every attempt should be made to combat the cardiac insufficiency as well as attempting to relieve the special symptoms. No special treatment can be given for dyspnœa or cyanosis other than the treatment of the cardiac insufficiency given above. Often the dyspnœa is due to a neglected hydrothorax or to an associated bronchitis, and these conditions should have their appropriate treatment. Oxygen has been suggested for the treatment of urgent dyspnœa, but will be found to be of little or no value. Gastric symptoms are often annoying—lack of appetite, nausea, flatulence. These symptoms are frequently due to a congestion of the viscera, and here small doses of dilute hydrochloric acid and pepsin—ten or fifteen minims of hydrochloric acid to a drachm of the essence of pepsin, taken three times a day—will often result in marked amelioration of these symptoms. Occasionally these symptoms may be due to digitalis or other drugs, and, if so, the offending drugs should be withdrawn. Cough is frequently very annoying and may be relieved by the use of codeine in one-quarter grain doses given every three or four hours; an additional dose at night will sometimes give the patient a comfortable night where otherwise the cough would keep him awake most of the night. Sometimes the cough is so persistent that a hypodermatic injection of morphine—one-eighth to one-quarter grain—is necessary.

*Sleeplessness.*—The milder sedatives are often all that is necessary to ensure a good night's rest: sodium bromide, twenty grains, with one-quarter to one-half a grain of codeine, or fifteen grains of trional, or five to ten grains of veronal. Veronal should not be used regularly, as some people are very susceptible to it, even when only given in five-grain doses, and after its continuous use unpleasant symptoms frequently arise. Chloral hydrate is of great value, especially in cases of hypertension with cardiac pain, when the adminis-

tration of two or three grains every three hours during the day or five to twenty grains at night will be found to be most beneficial.

In severe dyspnoea or with cardiac pain, and when marked discomfort is present, sometimes no sleep can be obtained without the use of morphine. This is given hypodermatically, and at first may be given only occasionally at night, but in advanced cases where sleeplessness and restlessness are so constant it may be necessary to increase the dose and to give it several times a day, and, in advanced and hopeless cases, it may even be necessary to allow the patient to form the opium habit under the direction of the physician.

Occasionally it may be found that morphine in doses of one-eighth of a grain in conjunction with tincture of digitalis may be administered with considerable relief to the patient.

*Venesection.*—With all our modern methods of treatment and with a large number of drugs and other measures at our command, the old-fashioned method of treatment by bleeding is too often forgotten. It is not infrequent, especially in hospital practice, to find a patient with cardiac insufficiency who has made some sudden exertion and has had a severe attack of heart-failure with marked dyspnoea and cyanosis, and with rapid and delirious heart-action. Here bleeding must be prompt and sufficient if the patient is to be saved. The withdrawal of from at least three hundred to six hundred cubic centimetres is necessary in an adult, in order to obtain relief from right-sided cardiac embarrassment. If œdema of the legs be not present a bandage may be placed around each thigh tightly enough to cause obstruction of the venous return. This also withholds a certain amount of venous blood from returning to the heart. These should not be left on over an hour. If the cyanosis and dyspnoea be not relieved a similar amount of blood may be withdrawn from another vein. This second bleeding may produce the desired effect.

*Edema of the Lungs.*—During the course of chronic endocarditis there may occur an attack of œdema of the lungs which may be a direct result of over-exercise, or a result of some accident such as an embolus, or infarct, or from an unexplained cause, or they may occur as a terminal event at the end of a prolonged and irremediable cardiac insufficiency. Œdema of the lungs should not be considered a hopeless condition and should be combated promptly and energetically. The injection, hypodermatically, of atropine, 0.01 grain,



and repeated till there is slight dilatation of the pupils, and persistent and urgent dry cups to the front and back of the chest for an hour, and repeated at intervals of half an hour for half an hour at a time, are of use. Venesection is indicated if the blood is dammed back in the lungs from an insufficiency of the right ventricle, as will be shown by the presence of marked cyanosis. These three measures, if promptly and energetically carried out, will often tide over the acute attack until such drugs as digitalis and caffeine, and so on, have an opportunity for a therapeutic effect.

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# MASSIVE INTRAMUSCULAR INJECTIONS OF DOUBLE HYDROCHLORIDE OF QUININE AND UREA IN THE TREATMENT OF PNEUMONIA<sup>1</sup>

WITH REMARKS ON INJECTION OF COCAINE HYDROCHLORIDE AS AN  
AUXILIARY MEASURE FOR THE SPECIAL PURPOSE OF  
RAISING BLOOD-PRESSURE

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GENTLEMEN: This man is brought in to illustrate the effect of the double hydrochloride of quinine and urea in the treatment of acute lobar pneumonia. The treatment of pneumonia by quinine is by no means new. It was old when I studied medicine. Indeed, there is a long-standing tradition among physicians that very large doses of this drug given at the inception of an attack of pneumonia will abort or "jugulate" the disease. An obvious objection has always been urged against that statement—although it was put forth by eminent clinical observers—namely, that if the disease does not develop one cannot be sure that the patient was going to have pneumonia. Therefore, he cannot be sure that he has stopped it. However, the tradition is one of those persistent things that will not down.

Very early in my own practice I had an experience which goes somewhat toward confirming it. I was called at about 2 o'clock one morning to a patient who had had a severe, sudden chill two hours earlier. When I saw him, the temperature was about 104° F., there was roughening of respiration, with a patch of mingled dulness and hyperresonance over the base of one lung, I forget which side, probably the left. There was pain, confined to the nipple. The man was coughing. His breathing was hurried but not difficult. Apparently it was the initial congestive stage, which we so seldom see, of acute lobar pneumonia. I prescribed quinine, ordering that 10 grains be given at once and repeated in six hours, thinking that we would let the

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<sup>1</sup> Clinical lecture delivered at the Philadelphia General Hospital, December 9, 1911.

patient sleep, if possible. His wife misunderstood the direction and gave him 10 grains every hour, so that he took 60 grains of quinine between 2 and 7 o'clock in the morning. I saw him at 8 o'clock perfectly comfortable and breathing quietly. The pulse had gone down, temperature had gone down. The lung, however, showed increased dulness; in one part crepitant râles, in another, bronchial breathing. In the course of four or five days the physical signs cleared up, but during those four or five days, while the physical signs in the lung persisted, there was no fever, respiration was normal and pulse practically normal. The significant fact is this: that here was a man who had taken 60 grains of quinine in five hours, but had not had the slightest cinchonism or ill effect of any kind. On the contrary, all his distressing symptoms had disappeared.

That, of course, reminded me of the old tradition that quinine aborts pneumonia, but still I did not feel like acting on the hint. I should have done so, I admit, but in the early days of my practice I was not bold enough to go ahead. Meanwhile Aufrecht and Petzold introduced the subcutaneous use of quinine in pneumonia; not in very large doses, however. About eight years ago Galbraith, of Texas, reported a large number of cases that he had treated with massive doses, about 75 to 100 grains daily, without a death. I think this report included 100 cases, and later he supplemented that with other cases. Galbraith's statistics have been disputed. He has been made an honorary member of the Ananias Club, and great discredit has been cast upon his work. Nevertheless, I am satisfied that it is a true and faithful record of experience. It recalled to me that early experience of mine of which I have told you; and in connection with the work of others which I had read of, and the tradition which I have mentioned, led me to adopt the systematic treatment of pneumonia with massive doses of quinine. My experience with the use of quinine and urea hydrochloride in malaria induced me, however, to use the latter drug in place of the particular quinine salt—bisulphate, I think—that Galbraith had used, and to administer it by intramuscular injection. I have since learned that Dr. F. P. Henry, of this hospital, had two years earlier been treating his cases of pneumonia here with hypodermic injections of quinine chlorhydrosulphate, though not in the massive doses that I have been using.

Now this patient is brought before you simply to illustrate the

result and the method of treatment. It is unnecessary to go into extended consideration of his case. He is a laborer, 38 years of age. He had a marked chill four days before admission, after which he became prostrated, and two days before admission he had two chills, increasing his distress. When he came in he had cough, rusty sputum, pain in the right side of the chest. He exhibited rapid respiration, frequent pulse, the ordinary physical signs of pneumonia in the stage of red hepatization; but, all in all, it was not a particularly severe case. The temperature was in the neighborhood of  $103^{\circ}$ , showing some tendency to go higher; the respirations were 50; the pulse 130; and the systolic blood-pressure 120. This last was the most important symptom. I will speak in a moment about the relation between the systolic blood-pressure and the pulse frequency.

In moderate cases of pneumonia much medication is not required. Usually, by exposing the patient to fresh air, out of doors if possible; sponging him, not to reduce the temperature, but simply for the purpose of stimulating the skin and keeping him clean; giving water to drink freely; feeding moderately, but not overloading the stomach—using a diet appropriate to the febrile condition, chiefly broth, soft-boiled eggs, milk, milk toast; seeing that he receives a sufficient number of calories as well as the minimum of proteids in an easily digestible form—and, especially, keeping the urine alkaline, one will put the patient in condition to recover. Such medicines as may be administered from time to time are to be chosen on symptomatic indications. But there are severe cases as well as moderate ones, and this “expectant plan” is not applicable in the severe cases.

As you know, about 70 to 75 per cent. of all cases of pneumonia get well, and it is said that about 25 to 30 per cent. will die, no matter what the treatment. These are the statistics of therapeutic nihilists. The only difference treatment makes, they say, is that under one plan Jack will die and Tom will get well; under another, Tom will get well and Jack will die. But I am not a therapeutic nihilist, and I cannot look upon a sick man simply as an item in a table of statistics. I believe we can save both Tom and Jack. At all events, we can try to do so, and by trying intelligently we can reduce very considerably that proportion of 25 or 30 per cent. of deaths in pneumonia. Certainly we can do so in private practice. We have done so, also, in Jefferson Hospital. Here in the Philadelphia Hos-



pital, it is true, there has been in some years an even larger percentage of deaths, but that is because we get here a very large proportion of terminal infections, old persons, alcoholics, persons who have been ill nourished and badly exposed, persons suffering with arteriosclerosis, and chronic heart and kidney lesions, patients admitted late in the development of the disease—probably the most unfavorable collection of cases that could be imagined. Some years the mortality has been as high as 50 per cent., and it is never expected to be less than 25 per cent., so that any reduction below that is to be credited to the treatment. And the treatment by quinine has had a mortality here during seven years of less than 20 per cent.

This man, although his case was not among the most severe, and although he would probably have gotten well under almost any form of rational treatment, was still a fit person to receive the quinine treatment, which I am using in a systematic way here, whenever applicable. We gave him an injection of the quinine and urea salt, 15 grains (one gramme), under the skin, and repeated it as often as his temperature rose above  $102^{\circ}$ . Just as we take a bathing line in typhoid fever, I take an injection line in the treatment of pneumonia with quinine, and for a convenient index I take about the same point ( $102^{\circ}$  F.). Not that the temperature alone is an indication for giving the drug. The treatment is not for the purpose of reducing the temperature (though it does that remarkably), but the temperature is an index of the general condition of the patient. It is a well-known fact that patients with acute lobar pneumonia do very much better at about a temperature of  $102^{\circ}$  than they do at a temperature of  $105^{\circ}$  or  $101^{\circ}$ . One doesn't—or shouldn't—try to reduce the temperature of his patient too low in pneumonia. Hippocrates long ago pointed out that fever—that is to say, fever-heat—is one of the natural defensive reactions. As we put it now, fever is part of the self-defence of the organism against infection. Hippocrates used a different form of statement, and our successors will probably express the same thought in still other terms. But the fact remains that the febrile reaction is part of the innate defence of the animal life against bacterial invasion, and that to interfere with that defence usually brings disaster rather than victory. It may, however, need regulation and control. So, taking  $102^{\circ}$  F. as our approximate line, we inject into a muscle 1 to 1.5 grammes (15 to 25 grains) of the

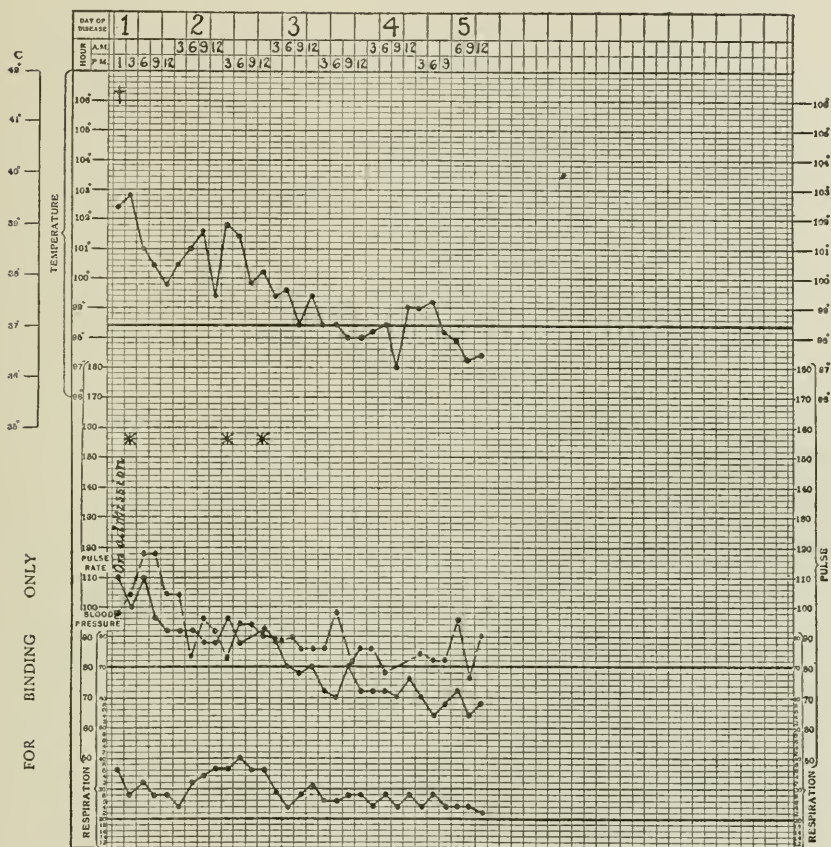
double hydrochloride of quinine and urea, and repeat every third hour or fourth hour, so long as the temperature tends to rise above 102°. We take the temperature and, if necessary, repeat the injection every three or four hours, according to indications and results. The dose stated is for a vigorous adult. Children and weak persons get proportionately less. So, too, the adult's initial dose is 25 grains or less, according to the severity of the case; and the successive doses are lessened, or the intervals prolonged, as the effects become more and more manifest. In that way in a large series of cases we have given amounts varying from 1 to 8 grammes (15 to 120 grains)—in one case 10 grammes (150 grains)—in the first 24 hours; and from 3 to 12 grammes (45 to 180 grains)—or even more—in the course of 48 to 72 hours. After that, smaller doses are continued by the mouth, if necessary—5 to 15 grains daily. The idea is to adapt the dose to the individual case; and to give the patient all the quinine that he needs, and can take without inducing quinine poisoning, in the first two or three days that he comes under observation. At first I administered the drug without fixing a temperature limit, simply giving it every three hours, so long as cinchonism was not developed, until a certain maximum quantity had been reached or the respiration had begun to drop down toward the normal line. But I have learned to prefer the other plan when I cannot keep in close personal touch with the case, and must leave directions with an interne—or, in private practice, with a nurse. In consulting practice, the attending physician is advised to leave matters "flexible" if he can be at hand, but to order the routine when he must trust to another.

In 192 cases that I summarized in a report presented to the Association of American Physicians last year the mortality was 12 per cent. This included a large number of unfavorable cases treated at this hospital, with a mortality of not quite 20 per cent. So the normal mortality, as we may call it, is certainly less than 10 per cent. under this plan of treatment; and if the cases could be seen early and the treatment thus begun promptly, it should be even less. Here it is rare to get a patient earlier than the third day.

A significant fact is that, despite the enormous doses of the most active quinine salt, and a method of administration almost as effective as intravenous injection, cinchonism does not develop. The temperature and the pulse-rate fall gradually and proportionately, but respira-

tion much more rapidly. The physical signs, however, of percussion and auscultation are not changed. Apparently they pass through the usual stages, unaffected by the treatment.

СЛАВЯН. I.



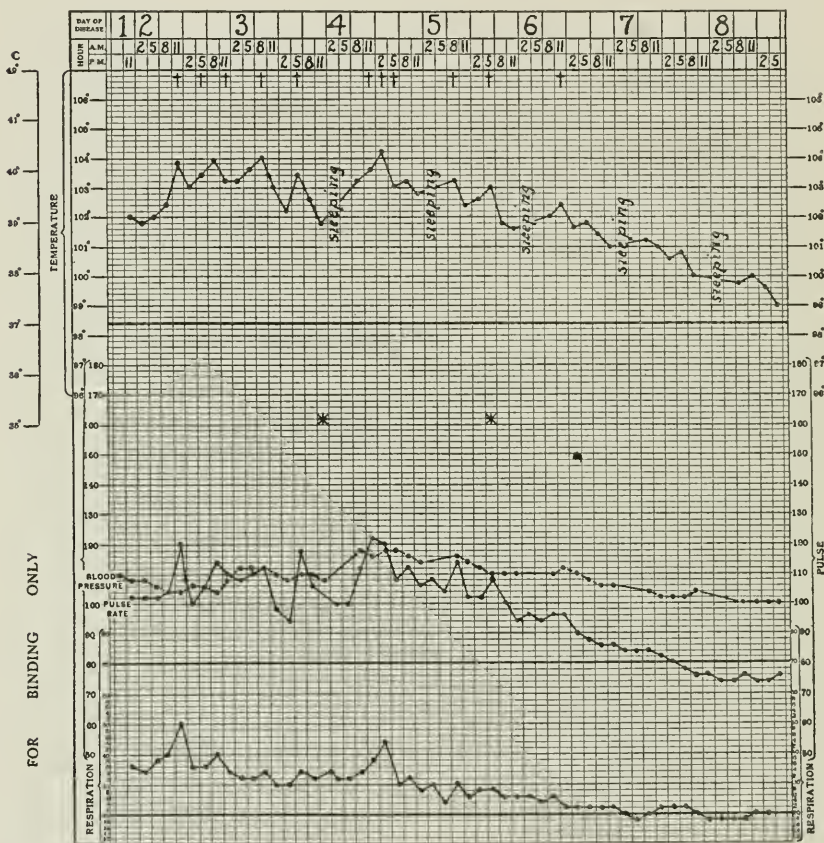
**Case Summary:** (Observation every third hour.) (*Acute lobar pneumonia; moderate severity; left lower lobe.*) B. F., male, aged 37 years. Pneumococci in sputum; chill; no vomiting; herpes; cyanosis; no delirium. White blood cells, 16,200. Polymorphonuclear, 75 per cent. †One injection of quinine and urea hydrochloride, 1 Gm., on admission, fourth day, two on fifth day, with resulting elevation of blood-pressure. Duration from chill to resolution, 8 days. No complication. Recovery.

You can see on some of these charts, that I am passing around, the rather rapid fall of the respiration. Here is a case, for example, where the man got ten injections of 15 grains each. His temperature kept up pretty steadily until he had the ninth injection,



but the respiration began to go down early. Here is another chart showing the same thing—early decline of respiration beyond pulse-rate. In fact, there is a general tendency to restore the normal

CHART II.

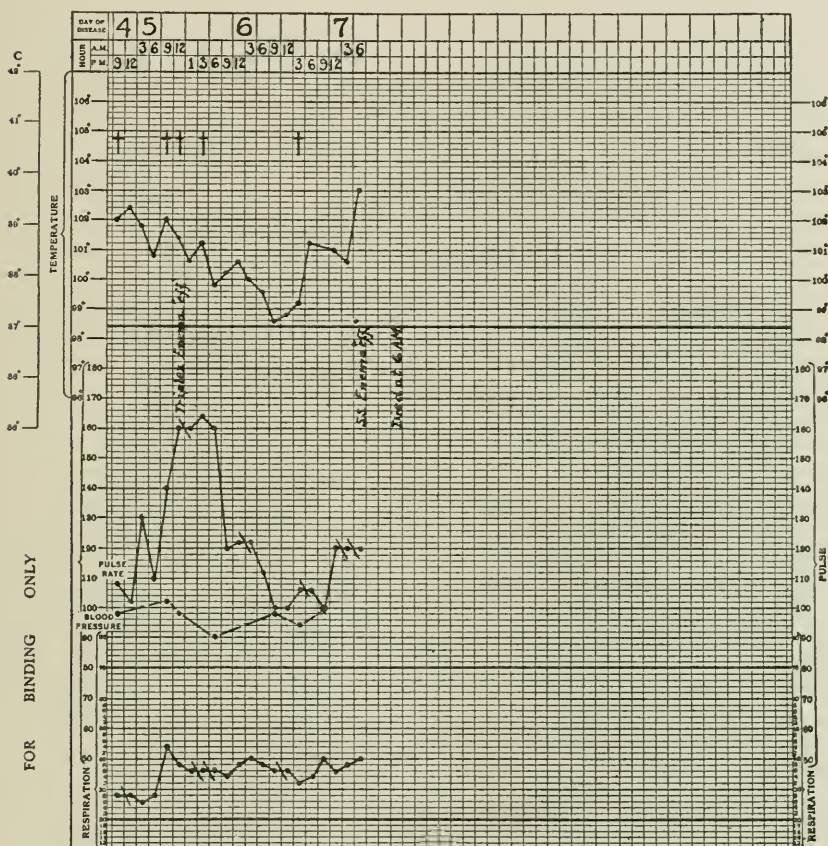


Case Summary: (Observations every 3 hours.) *Double apical pneumonia*. R. F., male, aged 48 years. Pneumococci abundant in sputum; chill; delirium; cyanosis; herpes. First injection on second day, last injection on sixth day, total quantity 11 Gm. in 75 hours. Rise of temperature on 4th day coincident with new involvement. Duration to complete resolution 14 days. Polymorphonuclear leucocytes increased by quinine. † Injection 1 Gm. quinine and urea hydrochloride (11 in all). \* Injection 0.03 Gm. cocain hydrochloride (2 in all).

pulse-respiration ratio; not to 1:4 always, but to less than 1:3. Observe, also, that the decline of temperature is by lysis—not crisis. Here is a case in which only one injection was needed (Chart I). Note again the decline by lysis instead of by crisis.

Note, also, the good effect of cocaine on blood-pressure. Of this I shall have something further to say later on. Here is a case of an alcoholic patient with delirium tremens. He got eleven injections,

CHART III.



Case Summary: *Acute double lobar pneumonia involving greater portion of both lungs.* Alcoholic patient admitted 4th day apparently moribund. Great subjective improvement under treatment, but pulse-rate continued to rise and pressure remained low. Blood culture shows pneumococci. † 5 injections quinine and urea hydrochloride, 1 Gm. each. First on fourth day, last on sixth day. No cinchonism. Death on seventh day. Autopsy showed complete want of resolution and diffuse suppuration in both lungs.

each of 15 grains of quinine and urea hydrochloride, which had a quieting effect (Chart II). You can see here the rapid decline of the respiration from 50 down to 30, and then to 25, and finally to normal, and a coincident decline of pulse and temperature. Here



is a pretty bad case in which recovery took place. There were two injections of 25 grains each and nine injections of 15 grains each in 72 hours; that is, 185 grains in all. And so I might multiply the charts. The important point to which I wish to call attention is this: that that amount of quinine might kill the patient if he did not need it. At all events, severe cinchonism would result from such large doses of quinine if there were not something in the patient's system which neutralized them. It is fair to suppose that the condition of the patient which prevents the quinine from poisoning him is the presence of some chemical substance which neutralizes the drug; and if that be the case then the drug must likewise neutralize that other and abnormal substance. Now a marked result of the injection of quinine is the increase of blood-pressure proportionately to the abnormal pulse frequency. Please observe that I do not say increase of blood-pressure over the normal height—but *an increase of blood-pressure proportionately to the abnormal pulse-rate*. Another effect is to decrease the rate and increase the ease of respiration. To these facts add another: The danger in pneumonia arises from a toxæmia which acts particularly upon the sympathetic (autonomic) nervous system and chiefly on the vasomotor apparatus, but to some extent on the heart and the respiratory centres, embarrassing and quickening the respiration, *increasing the pulse-rate*, and *depressing the blood-pressure*. Hence it is fair to conclude that the action of the quinine is antitoxic. At all events, whether it acts by a direct chemical neutralization or by some indirect method which we cannot make out as yet, not having sufficient data, it is antagonistic to the toxæmia and it overcomes the two most severe symptoms. One of these is a symptom of danger and the other is a cause of distress. The symptom of danger is the lowering of the blood-pressure and the distress is the labored respiration.

Now if you have ever seen a crisis in acute lobar pneumonia you will understand exactly what I mean. At one moment the patient is gasping for breath, not only breathing rapidly, but with great difficulty; he suffers acutely; his lips are blue, his cheeks have a dusky flush, his heart is beating rapidly and feebly; apparently he is near dissolution. An hour or two later, following a sudden fall of temperature, accompanied with profuse perspiration, the patient, if he has not succumbed to the critical disturbance, is breathing easily, his

heart is beating quietly and strongly, he is the picture of comfort. The ashen or cyanotic hue of his face has given way to a healthful, ruddy color, and altogether there has been a complete transformation. Yet if we examine the chest of this patient we find practically no difference. The dulness and the bronchial breathing are still there, showing that consolidation persists. The physical condition of the lungs is practically the same after the crisis as it was before. Perhaps the râles of resolution may be just beginning, but it will be several days before the consolidation disappears. And yet the patient is breathing comfortably. That is a conclusive proof that the difficulty of respiration in pneumonia is not dependent solely, or even chiefly, upon the tissue changes in the lung, but that it is caused in greatest measure by the toxæmia. In some cases, it is true—and these are usually fatal cases—the lung, or even both lungs, may be extensively, or perhaps almost entirely, consolidated; or there may be a diffuse suppuration. The crisis does not occur—or, if it does, death occurs with it. In such cases there is, in fact, in addition to the toxic disturbance, a mechanical obstruction to respiration. In the great majority of instances, however, the mechanical difficulty is relatively unimportant. At the crisis the toxæmia is overcome, perhaps by sudden liberation of antitoxins—but that is not known. However it comes about, the effect, in favorable cases, is to relieve the difficulty of breathing and to restore the tone and vigor of the circulation. Now the treatment by quinine seems to anticipate, and to bring about quietly and gradually—and *without the danger that ordinarily attends crisis*—the relief that otherwise comes late and critically. It prevents or mitigates the distress in respiration and it keeps up the blood-pressure, so that the patient has a chance to recover. Hence we are justified in regarding the effect—at least tentatively—as antitoxic. The structural pathological processes, however, go through their normal evolution, apparently unaffected. When we look at a large number of case-histories, resolution seems, on the whole, to be slightly delayed; but that may be only because recovery takes place in so many cases in which, under other treatment, there would have been no resolution at all—early or late.

I have spoken about the relation between the *pulse frequency* (as measured in beats per minute) and the *systolic blood-pressure* (measured in millimetres of mercury). I wish now to dwell upon this.

It is of great moment. Gibson, of Edinburgh, has called attention to the fact that when the figure representing blood-pressure, thus measured, falls below the figure representing the rate of the pulse, the patient is in danger. On the other hand, when it remains equal to, or above, the frequency of the pulse, the patient is likely to recover. In other words, excess of frequency over pressure is a bad prognostic omen; excess of pressure over frequency is a good prognostic omen. Also a pressure of less than 100 is a bad sign, independently of the pulse-rate. The rule is not absolute and invariable, but it is true of the great majority of cases, and the importance of it is that it gives a clue to the medication. We have not trusted to the quinine alone in the cases in which we have found this condition (in which the blood-pressure has been markedly or persistently below the pulse frequency or has shown a tendency to fall below 100 mm. Hg.), but we have given, in addition, some drug that would tend to heighten vascular tension—some drug having so-called “pressor” or “vaso-constrictive” action.

I have been looking for a chart showing an unfavorable result; one in which this “Gibson phenomenon” was present, and in which we were unable to get the blood-pressure up. Here it is (Chart III). The blood-pressure is measured in millimetres of mercury; the pulse frequency is measured in beats per minute. This chart, from Jefferson Hospital, shows a pulse-rate of 165 and a blood-pressure of only 100 on admission of the patient, about the fourth day of the disease. He was then nearly moribund. No treatment offered much hope, but I decided to give the man such chance as the quinine might afford. Of course, we gave strychnine, alcohol, and camphor also. He had been getting digitalis before admission to the hospital. I do not think we used cocaine in this case—but no drug would have changed the result. Oxygen gave ease. We were able to bring the pulse-rate down to a range between 100 and 120, but we could not increase the blood-pressure. It continued to fall, and we had the utmost difficulty in keeping it around 90. Although life was unexpectedly prolonged and the patient's comfort promoted, death occurred on the seventh day. At the autopsy it was found that both lungs were consolidated throughout, and that there was diffuse suppuration, so that recovery was not to have been expected.

Here, in marked contrast, is another chart. The case was one

of prolonged bronchopneumonia with pleurisy, admitted to Jefferson Hospital on the fourteenth day. The patient came in with a pulse-rate of nearly 140 and with a blood-pressure of 118. That was an unfavorable indication. Under treatment, however, the pulse-rate came down to about 100 and the blood-pressure ran up to 110 and finally to 120. The patient recovered. While this was not a case of lobar, but one of lobular pneumonia, the principle remains the same.

The two lines—systolic pressure and pulse frequency—being charted upon the same vertical, their distance gives to the eye at once this important relation. In the fatal case, there is the pulse frequency line at 160, and way below it, at 100, the blood-pressure. So long as the frequency line keeps above the pressure line it is an unfavorable omen. When we can get the two together, whether by bringing the higher one low or the lower one high, then we may say that we have a balanced condition. If we can get the pressure line to go up and at the same time the frequency line to come down, then we have a condition tending to recovery. If both go down and pressure still keeps below frequency, that is bad. If both go up and frequency still keeps above pressure, that is also bad. What we want to do is to bring the frequency low and send the pressure high, and if we can thus reverse the relation, our patient will probably get well.\*

The importance of this is that this "Gibson ratio" is our guide for stimulation and for the administration of adrenalin, pituitrin, strychnine, digitalis, camphor, caffeine, cocaine, or whatever may be the drug best suited to bring about recovery in that particular individual. My own favorites are camphor, 30 minims (2 c.c.) of a 10 per cent. solution in sterilized olive oil, hypodermically, repeated as necessary; and cocaine hydrochloride  $\frac{1}{2}$  grain (0.03 gramme) hypodermically, repeated as necessary. I have given it every third hour for eight or more doses; or in some cases only once or twice in the course of treatment. Chart II shows the good effect of cocaine in bringing up the pressure, and here is another demonstration from

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\* Normally the ratio of pulse frequency to pressure (measured as stated) is approximately 72 to 135 or 140; say 1:2. In pneumonia it may become instead 150 to 100; say 1.5:1. If by treatment it can be made 1:1 the condition is better but still dubious; if it can be made 1:1.2 or more, the condition is favorable.



quite a severe case of double pneumonia. The patient was admitted to Jefferson Hospital on the third day, the right lung only being involved. He received one injection of the quinine salt and was apparently on the road to recovery, when the renewal of high temperature and rapid respiration, with increase of pulse-rate and drop in blood-pressure, gave signal of danger, and it was found that consolidation had appeared in the left lung. Injections were resumed and ten, indeed, were given before recovery was assured. Cocaine was given twice, each time with prompt resulting benefit.

Now I am not advocating the use of quinine, or of quinine and cocaine, as a specific treatment in acute lobar pneumonia. I am only telling you what it does; showing you in how far, and why, according to my judgment, it assists the patient to recover. If you should ask me what is the principal and most necessary detail of treatment, I would say it is the exposure of the patient to fresh air—on the roof, or on a porch, or in a tent, if possible; if not, in a big room with all the windows and doors open and the heat excluded. We need not only fresh air, but cold air—somewhere between 30° and 50° F. Tell the objecting friends that the patient can't "catch cold," but that by staying indoors he may "catch hot." The patient's body, however, must be kept warm by coverings and by "hot bottles," "warming pans," etc. Moreover, I am one of those who advocate the application of external heat, rather than ice bags or cold compresses, to the chest. It may be applied by means of an electric warming pad, by flaxseed or other poultice, by lamb's wool or cotton "wadding" jacket, or by whatever method may be most available. I have already spoken of the food, of the stimulation as necessary, and of the symptomatic medication.

In those cases in which there is considerable consolidation, in which there is not only toxic dyspnoea but also the mechanical difficulty of breathing, the inhalation of oxygen should be carried out, and carried out persistently. The reason that so many failures are made with the administration of oxygen is that most physicians only play with it. They have the patient inhale oxygen for five or ten minutes, three or four times a day. One might as well put the oxygen tube out of the window as into the patient's mouth for that period of time. To save a patient's life by inhalation of oxygen, we must give it continuously and freely. It costs money—it wastes



oxygen. But if the oxygen is to save life, that is the only way in which it can be done. It is only in those cases in which there is actual obstruction, mechanical dyspnoea on account of the extreme consolidation of the lung, that oxygen is needed.

In addition, we should keep the blood—as shown by the urine—alkaline. We can do that with ammonium preparations or with sodium or potassium preparations, or with a mixture of compounds of different alkaline bases—solution of ammonium acetate, or potassium citrate, or sodium bicarbonate, or ammonium carbonate, or ammonium chloride, associated or alternated—whatever you please. Also be sure that the patient drinks and passes a large quantity of water. The alkaline draughts are also good diuretics; the ammonium preparations stimulating expectorants as well. Of course, the bowels must be kept open, and all other details of good nursing looked to.

Saline infusion under the skin was introduced some twenty-odd years ago by my friend, Dr. F. P. Henry, now senior physician to this hospital. This I advise in every case which shows a severe toxæmia, or every case in which the chlorides are more than ordinarily diminished in the urine, or in which the quantity of the urine is scanty. It can do no harm in any case.

Now the combination of fresh air, alkalines, quinine in large doses under the skin (and preferably this particular quinine and urea salt, of which I am speaking), proper food, and appropriate stimulation—alcohol, cocaine, strychnine, atropine, etc., when called for by the excess of pulse-rate over blood-pressure or by other symptoms—also saline infusion and oxygen when needed—will, I think, save many patients that might otherwise die.

In advocating this treatment I do not oppose the treatment by bacterial products. I believe that treatment by bacterins (vaccination, as it is improperly called) is extremely valuable; and in my own wards here and at Jefferson Hospital we have had excellent results from a somewhat limited resort to it. But it has not yet been sufficiently developed for me to be in position to tell students just when and how they ought to use it. I have great faith in the treatment by mixed bacterins in suitable cases. Shafer's results with bacterial, or rather toxic, filtrates are well worthy of attention also; though I have no faith in that or any other treatment—even quinine—as a routine in every case. The wise physician modifies the treat-

ment to suit the case and the patient. This applies to quinine as to everything else. But I know nothing else both so valuable and so readily available in the great majority of cases of pneumonia as this which I have been describing.

The advantages of quinine and urea hydrochloride over other quinine salts are its great solubility and its apparent higher efficacy. The objection brought against its hypodermic use is the frequent occurrence of cellulitis, abscess, or slough. This may be averted by observing a few simple precautions. In the first place, all the instruments to be used are sterilized by boiling. The skin over the part into which the injection is to be made is cleansed with green soap and water; and then an area of some two inches in diameter is painted with tincture of iodine. A high-pressure syringe is used, and the long needle attached thereto is plunged deeply into the muscle. Care is taken to expel all the contents of the syringe, so that in its withdrawal none of the solution shall be dropped on the surrounding tissue. The puncture is sealed with iodoform-collodion. Or a piece of rubber tissue that has been dipped in bichloride solution may be stretched over the skin and the puncture made through this protective, and sealed with plain collodion. In more than twenty years I have had no worse accident than a transient swelling, when the precautionary routine has been followed.

We use, by preference, a 50 per cent. solution of the drugs in hot sterile water; but when the capacity of the syringe available is less than 2 c.c., a stronger solution may be used. One simply dissolves the dose of the quinine salt in as much hot water as the syringe will hold. Hot water will take up nearly its own weight of the drug; thus 15 grains will dissolve readily in 20 minims (or even less) of hot water. I have not seen any local harm from the concentrated solution when due care has been observed in accordance with the directions just given. The site of injection may be arm, back, thigh, buttock, or any convenient place where there is sufficient muscular tissue.

## DECEPTIVE ONSET IN LOBAR PNEUMONIA

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THE ordinary classical case of lobar pneumonia as it occurs in hospital practice and in text-books, beginning suddenly in an apparently sound adult with chill, pain in the side, cough, headache, rapid respiration, and high temperature, forms a clear-cut picture that is easily grasped by the student and is pathognomonic of the disease. Such a history represents 60 per cent. of the cases seen in the wards of any large general hospital, the clientele of such institutions being drawn, for the most part, from adults. This syndrome, however, occurs only in the minority of cases seen in private practice, and here the clientele is largely made up from both extremes of life and adult females. Certainly not more than 40 per cent. of the cases seen in private practice in this city will present the classical picture. In less-congested districts it is apparently more common to find that the onset corresponds more closely to the text-book picture.

That the pneumonia actually begins at the time the patient experiences the chill and stitch in the side is not in accordance with the history of an incubation period, such as is the rule in all microbic diseases. During this incubation period there must necessarily be some symptoms. One often hears the statement that the patient never felt better in his life for a day or two before the pneumonia. This marked feeling of mental and physical exhilaration should of itself be viewed with suspicion. One must remember that not all toxins are depressant, that some of them have a strychnine-like action in some individuals, and this diversity in physiological effect may account for the stimulation.

In cases not associated with this feeling of exhilaration before the onset we generally find the patient complaining of more or less indefinite signs and groups of symptoms which, unless we are on our guard, will lead us to diagnosticate an entirely different ailment. These early symptoms are often quite brusque and severe, appearing

in some other portion of the body, perhaps far removed from the lung, and seem to have no possible connection with anything in the chest. While of themselves they may not be pathognomonic of any disease, still they suggest a complaint in that particular location. Our minds are so constituted that this fixed notion of the location of disease resulting from the impression made by the brusque symptoms at the start prevents the interposition of a second picture showing the possibility of the true disease in the background.

If we could approach each and every case methodically, by taking observations and collecting them, and then making our diagnosis, we would err less frequently. The exigencies of practice make this an evanescent dream, and most of our diagnoses are made within the first five minutes of the examination. This failure to properly suppress the significance of the initial brusque symptoms, as they are impressed on the mind of the observer, explains why one who comes into the case later, being free from prejudices, if I may use the term, will often make a satisfactory diagnosis. The clinical picture that presents itself in the majority of cases, as stated above, falls within one of several groups, and many cases have little that suggest pulmonary involvement.

It is at both ends of life that we are apt to see greatest variations in the mode of onset, and consequently pneumonia is, at these periods, more apt to be mistaken for something else. In children the clinical history is often as follows: a well-nourished child has been peevish or restless for two or three days, with gradual loss of appetite, although liquids are readily taken. The examination shows a temperature of  $104^{\circ}$ , pulse 130, respirations 30 to 40. The child cries and is restless, the chest examination is negative, the tongue is coated, there has been vomiting and oftentimes a slight diarrhœa. Under such conditions we are apt to think that we are dealing with a digestive disorder, so very common at this age. If the condition has lasted a week or more, typhoid fever, of course, is suspected, especially when typhoid is endemic. This is further strengthened by the fact that marked cough is often a late symptom. If the fever persists in spite of castor oil, if there is nothing found in the throat or ears, we are justified, in the vast majority of cases, in making a diagnosis of central pneumonia. This is rendered almost certain if there be cough with an occasional râle. It is true that other



infections like tubercular meningitis and poliomyelitis do begin this way, but they are so rare, as compared with the incidence of pneumonia, that they are negligible in arriving at a conclusion. The disturbed pulse and respiration ratio is not always a marked feature in the beginning of such pneumonia, although later it becomes very apparent. Probably most cases that one sees in children begin with the above symptoms, plus a head cold and bronchitis, and are termed grippé. There is often a question whether the bronchitis is a preliminary, or is itself an accompanying symptom of the pneumonia. I do not refer, of course, to cases of bronchopneumonia that always begin after a bronchitis; the consolidation, if looked for, occurring sometimes so soon after the initial symptoms as to make it seem as though they were the same infection.

Another type of case that one sees almost exclusively in childhood is about as follows: A child of three years of age is taken suddenly ill after an indigestible meal, or perhaps in the middle of the night, with high temperature, restlessness, twitching at the slightest cause, or mayhap a convulsion. There is soon some retraction of the head and vomiting, the pupils may be dilated, with a temporary squint and photophobia; delirium is frequently present, although drowsiness is much more common; this verges at times almost into coma. Slight cough is not noticed; dyspnœa, when present, is ascribed to the pyrexia and the restlessness. The irregular breathing suggests the cerebral trouble as much as the other symptoms. Many physicians, on being introduced to this syndrome, achieve a great reputation for the cure of meningitis. These cases will, on the third or fourth day, show, in the vast majority of instances, a marked consolidation posteriorly, often at the apex. Happy is he who has the confidence of the family sufficient to prevent the suggestion of a consultant or a change of physicians. Inasmuch as 95 per cent. of the cases get well anyhow, probably no great harm has been done by the error. Upwards of 25 per cent. of the pneumonias of children show at the beginning some of the cerebral disturbances as outlined above.

Occasionally one sees a young patient with tonsillitis, headache, flushed face, white spots on the tonsils, and within 48 hours a well-marked pneumonia can be made out. In a few such instances pneumococci have been isolated from the throat. Another rare form of onset is with earache; a slight redness of the drum suggests that



temperature, pain, etc., are due to an otitis. Within a day or two careful routine examination reveals a pneumonia. These cases, with sharp onset of vomiting, headache, and dry skin, and a notable congestion of the pharynx, with a very much coated tongue, may resemble scarlet fever, and, especially if the child has been exposed to it, are very suggestive. A day or two is sufficient to rule out that disease.

Possibly the syndrome, the mistaking of which is of the greatest import, occurs usually in children from 5 to 12 years of age, although it does sometimes occur in adults. The patient is taken ill rather suddenly in the midst of play, with a pain in the right side of abdomen, vomits and can retain nothing. The temperature is  $104^{\circ}$ , pulse 120, respiration 40; the abdomen is tense, tender, and sometimes distended, there is more rigidity on the right side, and point-pressure is often present over the appendiceal region. The legs are drawn up, the eyes are sunken, and often there is an anxious expression on the face, and there is generally some headache. In some of these cases there will be a preliminary history of vague pains in the abdomen for a few days preceding the onset. A high leucocyte count goes far toward making a clinical picture of appendicitis. The irregular and rapid respiration is accounted for, in the mind of the attending physician, by the abdominal pain, and because of the evident distress of the child he neglects to sit him up and examine the back, because it will add to the suffering, and, as there are no signs pointing to the chest, he foregoes the examination. The second or third day may show the consolidation and clear up the diagnosis.

Many of the most eminent surgeons have been deceived repeatedly by this syndrome, and have operated only to find a healthy appendix or one with a slight "catarrh." When this happens to the leaders, how many times has it been done by those of less experience—when in these days even the recent graduate, without even a hospital experience, stands ready to attack the appendix of anyone the instant there is pain over McBurney's point associated with tenderness, temperature, and vomiting! Teachers would do well to impress on students that the "P" in appendicitis may mean the "P" in pneumonia in any case, but most especially in the young.

The abdominal symptoms that occur in childhood are sometimes duplicated in the adult. The pain and tenderness are apt to be a

little higher up on the abdomen and suggest acute gall-bladder trouble or, occasionally, a perforated gastric ulcer. This is especially so if the patient has had gastric symptoms for some time previously. And how many are there in these days who on close questioning will not give a history of gastric disorder? Fortunately this mistake is not so often made as is the case with appendicitis.

How many an aged man who appeared to be in his normal health, with the exception of a tired feeling that he credited to his advanced years, has gone to bed, sometimes without any medical attention, or perhaps after taking a simple mixture of his own selection, that of the corner druggist, or mayhap of some unfortunate physician, and has been found dead in bed the next day! Immediately the cry is raised that he has been doped, poisoned, or what not. When the postmortem is made it shows an entirely unsuspected pneumonia. Many of these patients, while not alcoholic, stifle the pain or discomfort by occasional drinks of whiskey and appear to be in their normal health until stricken in bed or elsewhere. The record of the coroner's office shows many such cases; few are diagnosticated as pneumonia, in most instances the classification being heart-failure.

The more common syndrome among the aged is as follows: For the past two or three days the patient has been complaining of lassitude and does not feel like getting around; he is easily tired and prefers to lie down. On examination the surface of the body feels so cool to the hand of the examiner that, unless the temperature be taken, he is apt to be deceived by the idea that the patient is free from temperature or fever. When taken by rectum it often does not go above  $100.5^{\circ}$  for two or three days. By mouth it may show only  $99^{\circ}$ ; there is no more cough than the patient has had for years. He has become so accustomed to his "catarrh," as he terms it, that he deems it only natural and physiological. Unless stimulants have been freely administered, the prostration is soon the most marked feature of the case. There is a quick but not necessarily rapid pulse of about 80 or 90 and respirations of 30 or thereabout. The neophyte may fail to take into account the fact that toxins as well as drugs are not so active in depressing the vagus at this time of life, and that such a pulse will correspond to one of 110 in a young adult. The respiration does not seem to be markedly quick, unless the patient makes an exertion. As a matter of fact, it is always quickened, but

the excursions are so slight that they are almost imperceptible. Even in well-marked cases the expectoration is slight and seldom bloody. Chilly sensations are complained of, which may be attributed to the usual easy susceptibility to feeling cold among the aged.

A careful examination of the face will always reveal a marked change in expression. There is some mental depression and a "don't care" spirit about the patient; there is loss of appetite and the tongue is always coated if the weakness has lasted 48 hours or more. This loss of appetite and coated tongue the patient often ascribes to disturbed digestion from some food that did not agree with him. Occasionally there is diarrhoea, and this seems to fill out the picture of disturbed digestion.

Because of the prostration and evident distress on exertion, as well as the resigned look of the patient and his friends, the physician desists from sitting the patient up and making a thorough examination of the chest and back, but lets the diagnosis go as malaria, grippe, typhoid, or the ready-made diagnosis of the patient as outlined above. On the third or fourth day the pneumonia is plain to every one by reason of the very rapid breathing, and is usually followed by the death of the patient.

In some of these cases pain is altogether absent, while in not a few aching pains in the bones may be complained of in the back and legs. The severity of these pains may vary from an occasional ache to pains of a severity that cause the patient to cry out at every movement. Often, with all of these aches, there may be no pain whatsoever in the chest. After a few days the chest pain almost always becomes noticeable.

Another rather common form of onset that is often met with in young strong adults is as follows: The patient is taken with a rather sudden illness, with headache, aching limbs associated with high temperature, lack of appetite, and possibly with a slight cough. This is almost always ascribed to influenza, especially if there be an epidemic of the disease in the neighborhood at the time. On the third or fourth day well-marked signs of consolidation are found, and the practitioner consoles himself by saying that the pneumonia is secondary to the influenza. It undoubtedly does follow the influenza in many cases, but the brusque and severe onset and the rapid appearance of the physical signs speak for a pneumococcus infection from

the beginning. Without cough one is apt to neglect the daily examination of the chest in such a case and miss the early physical signs in the chest.

Another group of cases in adults also in the prime of life presents the following picture: The patient complains of a multitude of symptoms of indefinite character lasting over a week or more; he will have had a headache, backache, lack of appetite, constipation, slight chilly feeling, and is tired all of the time. There is a temperature of  $101^{\circ}$ , pulse 90, respiration 30. This is, of course, very suggestive of typhoid fever to the one who is dealing with typhoid cases at that time, and malaria in malarial regions, the elder physicians often straddling by calling it typhoid malaria. A sharp pain in the side and some distress in breathing may be the first thing that will call the physician's attention to the fact that the lungs are already markedly consolidated. This will often be the tenth or eleventh day of the disease, and typhoid pneumonia saves the face of the physician.

A sallow-faced man of spare build who will give a history of not feeling just like himself for a couple of weeks goes to bed suffering from a cold in the head and chest. During the night he has one or more severe hemorrhages from the lungs, amounting to a cupful or more. The physician who is called will, in the vast majority of cases, diagnosticate pulmonary tuberculosis on the spot. In some of these cases, however, the temperature will be markedly elevated, and often there will be an increase in the number of respirations, which will be credited to the pulmonary tuberculosis and the apprehension. It is true that such cases may have possibly a tubercular focus, but so many of them recover without any physical sign being left that we should be careful of making such a diagnosis.

Recent researches seem to show that most cases of hæmoptysis in consumption are associated with the pneumococcus, so that possibly we have to deal with a small pneumonia at the same time in all cases of hæmophthisis.

When pneumonia occurs in the course of another disease the onset is frequently overlooked because the symptoms are not marked; this is especially so in serious diseases where there has been much prostration. The increased temperature is ascribed to an exacerbation of the underlying malady.



In typhoid fever, in the second or third week, unless an accurate chart is kept, the increased respiration and cough will escape notice, and until the dyspnœa becomes very marked there will be no suspicion of pneumonia. In these cases pain is seldom present, whereas in pulmonary tuberculosis marked pain and dyspnœa are evident at the onset, although there may be few other symptoms.

When seen in the protracted cases of ileocolitis, usually in children, the cough, pain, and dyspnœa are slight or entirely wanting; a slight acceleration of respiration with an increased temperature is the only suggestive sign and this is usually considered to indicate an exacerbation of the primary disease.

In advanced cancer a depression of spirits and slight increase of pulse, temperature, and respiration occur, and are usually ascribed to one of the septic consequences of the cancer. In a fair proportion of cases pneumonia is found.

A patient who has been a sufferer from chronic bronchitis and emphysema for years will complain of some tightness of the chest; on examination the rectal temperature will be  $101^{\circ}$ , pulse 100, respiration 34. There will be some dyspnœa, but this is credited to the emphysema. Careful examination reveals no chest signs except those that have been noticed months before on previous occasions—the cough and expectoration are markedly less than formerly. Typhoid fever is often suspected in such a case, and it may be four or five days before the bloody expectoration suggests the true disease.

Cases like the following are a frequent cause of discomfiture to the young house physician when the autopsy reveals the error. The family practitioner is generally spared the loss of his *amour propre* because there is no autopsy.

A young robust adult of alcoholic habit, as a result of an accident develops delirium tremens and dies after two or three days. During life there were no symptoms pointing to his lungs. At first there is a little trembling of the hands and tongue, and then delirium gets gradually worse. It is a busy delirium, and he has especial fear of black objects. He constantly wishes to get out of bed, and when lifted up he falls back like a stone.

The respirations were accelerated, it is true, but not more frequent than one would expect from a patient suffering from any severe delirium or intense excitement and restlessness. There was no marked



cough nor expectoration, and any slight cough was ascribed to the mucous catarrh of alcoholism. Possibly the urine would show chloride changes, but so many changes are apt to be found in the urine of a delirium tremens case that no account was taken of it. The patient dies suddenly and the autopsy reveals, in a very large proportion of cases, a lobar pneumonia. The same picture is of frequent occurrence in the maniacal insane. There are rare cases that appear at first as typical cases of inflammatory rheumatism and of gout, but the chest symptoms soon become quite marked and there is rarely any trouble in the diagnosis.

The cases that come on in the course of grippe give a history somewhat as follows: There has been an irregular and intermittent fever of moderate intensity, a severe inflammation of the nose and laryngitis, and a general bronchitis with considerable dyspnoea. From the fourth to the sixth day, or even later, there may be repeated shivering and a slight increase in temperature; the pain in the side, if there is any—and it is usually absent—is ascribed to exacerbation of the primary ailment. The expectoration is at first simply mucous or mucopurulent, and often may not change during the whole course of the disease. As the patient has been ill now for a couple of weeks one begins to question the possibility of general tuberculosis. In most cases, after the third day of the secondary disease or the tenth from the start the practitioner is awakened by the sight of rusty sputum and the increased respiration rate.

There are, no doubt, many other conditions that we are apt to mistake for pneumonia, but they are of uncommon occurrence, and it is not the purpose of this paper to give a tabulated list of such diseases, but rather to point out that the time-honored teaching that the onset of pneumonia is clear and sharply cut does not hold true for the pneumonia that one sees in the metropolitan centres at this time. It may well be that in the past they were different, but diseases change both in frequency and character, and lobar pneumonia is no exception to the rule.

## THE DIAGNOSIS AND TREATMENT OF LOCOMOTOR ATAXIA \*

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GENTLEMEN: When I uncover the knees of this patient you observe that his right knee-joint region is the seat of an enormous swelling and that there is nothing visibly abnormal in the left. You notice that the swelling is globular and the skin over it seems tense; yet there are no distended veins such as one might expect to see if one were dealing with a rapidly-growing osteosarcoma; there is neither the redness nor discoloration of an acute inflammatory condition; and there is no sinus nor other external evidence of a tuberculous affection. Were it an acute inflammatory condition with effusion into the knee-joint, you know the limb would assume the position of the greatest ease; the position in which least pressure would be exerted upon the inflamed articular nerves; the position in which the effused fluid would be under the minimum tension; the position, therefore, in which the synovial cavity would have the greatest holding capacity. This position clinical experience and experimental research have shown to be semiflexion of the knee-joint. Yet you see, in spite of the swelling this patient lies with his right leg fully extended. If we ask the patient to move his leg you notice that even with this enormous swelling his movements are apparently painless and of remarkably good range. But you perhaps further observe that as he bends his knee a broad furrow appears in the middle of the tumor and the distention increases on either side. The furrow coincides with the position and direction of the patellar tendon. The pressure of the tightened tendon has imposed a new form upon the swelling. The swelling consists, therefore, of fluid.

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From its shape, from the extent to which it passes up in front of the femur, and from its behavior on movement of the limb it must consist of fluid distending the capsule of the knee-joint. From the attitude of the limb, as we have already decided, the fluid does not arise from an acute inflammatory process. But, even in the absence of all inflammation, these sudden local alterations in the distention which we saw when the limb was bent could painlessly occur only in absence of the normal sensibility of the joint. So we may confidently deduce, merely from what we have observed, that we are here dealing with a disease in which effusion into a joint is associated with impairment of the sensibility of the deep structures.

On palpating the swelling I find its temperature is slightly higher than that of the other knee, but so little is the difference that it is probably referable to the heat-conducting properties of the effusion. Fluctuation on tapping is visible, and the patella requires to be depressed almost an inch before it reaches the femur. There is no thickening of the synovial membrane nor of the bones; and the relation of the bony landmarks of the joint is normal. You see I can move the leg freely and painlessly in every direction. You remember that the knee-joint depends for its integrity upon the muscle tendons which surround it, and in the extended position of the limb they allow practically no lateral movement. A considerable degree of lateral movement is present here. I can easily bend the leg both inwards and outwards. This abnormal mobility may be due to the stretching of the joint ligaments and tendons by the effusion. But it is also present in the left, the seemingly normal knee. And observe, if I abduct his lower limbs he can readily be made to assume attitudes which could be rivalled only by rachitic infants or by those ladies who in music halls softly subside into the posture technically termed the "splits." The patient, therefore, shows marked hypotonus.

A patient with such a silent, enormous knee effusion, with loss of deep sensibility, and with hypotonus is surely suffering from locomotor ataxia. On now examining him from a neurological standpoint we find his deep reflexes are absent; he has early optic atrophy in both eyes; his pupils are small and do not contract to light; he has diminished sensibility to the prick of a pin over almost his

whole body; pressing on his eyeballs does not make him wince; I apply severe pressure on his face and it does not hurt him, so he has lost not only the sensory fibres of his seventh nerve, but also the cruder pressure perception fibres which travel in the sympathetic. All over the body this loss of deep pressure pain can be demonstrated. Where normally one feels pain on pressure of a few kilos he remains unaffected by pressures of eight, nine, and ten kilos. Hence the painlessness of his knee, which is an example of one form of Charcot joint.

The patient gives the classical history of slight venereal infection in youth, followed in early middle age by the onset of difficulty in walking and lightning pains. The swelling of the joint appeared suddenly several years ago. It subsides somewhat on rest, but it troubles him so little that he persists in walking. And, as you now see, he, with the help of his stick, walks with remarkable facility.

The next patient was until four years ago prominent in business circles in his town. He then fell down an open shaft and suffered somewhat from shock, for, although his physical injuries appear to have been trivial, he was confined to bed for several days. Within a month of his accident he felt his feet heavy and numb and his legs dragging. He was said then to be suffering from traumatic neurasthenia, but he has not improved. And now, in addition to the numb and dragging sensations in his legs, he complains of a sinking feeling in the pit of his stomach "as if a heavy weight were pulling it down." His pupils are sluggish in daylight, although they move fairly briskly when exposed to bright light in a dark room; the left is more sluggish than the right, and the contraction is almost instantly followed by dilatation. The temporal halves of his discs are pale, but there is no marked diminution of either his visual acuity or of his visual fields. He has no affection of his other cranial nerves. His motor power is excellent in strength and range. You observed that he walked in without difficulty, that there was no striking alteration in his gait. He can unerringly touch the tip of his nose with the point of his finger; but, as he tells you, he is greatly practised in this manœuvre. By making him bring the points of his two indices together we can, however, demonstrate that he has a considerable loss of coördination in his upper limbs. And, although he walks apparently well, if we now ask him

to advance by placing in turn the heel of one foot in front of the toes of the other you see at once how he sways and tends to fall. His incoördination is markedly increased when he closes his eyes; and he shows, among other evidences of this, what is called Romberg's sign; *i.e.*, he cannot, with his eyes shut and with his feet close together, stand upright without markedly swaying. His right knee-jerk is absent; his left is present only on reinforcement; and his ankle-jerks are both lost. On stroking the sole of either foot you see the toes bend down as in the normal response. But observe how intolerant he is of scratching; how much he resents it; how energetically he withdraws his foot. The only loss of sensibility one can detect on his skin is an area over the front of his chest. This area extends roughly from the second to the seventh intercostal space. The loss in it is slight to cotton wool, conspicuous to the pain of pin prick; yet the analgesia is not absolute. He can tolerate very severe pressure over his muscles, bones, eyes, and testicles without wincing. But even where his sensibility to touch, to pin prick, and to deep pressure is impaired you see he still remains curiously sensitive to scratching.

You remember at our last meeting you saw several women suffering from alcoholic neuritis. In them the knee-jerks were absent, and over the feet there was loss of sensibility to cotton-wool touch and to pin prick. But you also remember how intolerant they all were of the slightest pressure; even light pressure apparently caused them pain. In cases of chronic alcoholism you sometimes get sluggish pupils; often loss of deep reflexes; but never diminished tenderness on pressure. A loss of tenderness on pressure is a valuable sign which in cases of doubt will help you to distinguish incipient locomotor ataxia from peripheral neuritis.

The reaction of the pupils, the cuirass of analgesia, the general diminution of sensibility to pain on pressure, the absent deep reflexes, and the incoördination tell us this patient is undoubtedly suffering from early locomotor ataxia. He gives a history of only one venereal infection, a slight attack of gonorrhœa at the age of eighteen. He married; his wife conceived thrice, and the three children are alive and well. His Wassermann reaction is negative and his cerebrospinal fluid shows no increase of cells. He was very anxious to try an injection of "606." I told him I thought it was



quite useless for him; but everything else had been tried in vain and he fretted that he could not get this much-lauded specific. So I yielded against my better judgment; for with such a low cell-count, and with the absence in him of any signs of an exudative process, I did not see what "606" could do. He had diarrhœa and headache for forty-eight hours after the injection. For two days afterwards he proclaimed he was well; all his troubles had disappeared; and then the sinking sensation in his stomach returned. But for two days the faith of this poor fellow in the drug, reinforced by the rubric of an intravenous injection and by the proof of the potency of the stuff in his headache and diarrhœa, enabled him to persuade himself that his trouble was at an end.

I have observed several remarkable instances of the psychic effect of "606" in cases of advanced organic nervous disease. Within a time so short as to preclude any but a psychic action, and without any change in the objective signs of the disease, the bedridden have stood,—attempted to walk,—and have continued to feel improvement even for several weeks. But sooner or later within the first month after the injection they relapse with dramatic suddenness. In other cases, where the physical signs indicate that the lesion is due mainly to specific vascular changes, no tangible improvement follows. But in disability arising from syphilitic meningeal thickening the improvement demonstrable even in the objective signs of the disease is often remarkable. You have seen in the wards five such cases. One of them relapsed nine weeks after his first injection, but he again began to improve after a second dose. The injection certainly sometimes produces powerful general effects, even when only forty centigrammes in very dilute solution (1200 C.c.) are administered; and you all saw the alarming local reaction which it produced in C.'s arm. Yet, among the score of patients so treated whom you have seen, there is none but wishes to have another dose.

The latest practice is to combine the injections of "606" with a course of mercurial treatment. The combined effect should ensure the speed of the "606" action with the permanence of the mercurial. But with the introduction of the joint medication will end the prospect of a final settlement of the claims of the new drug. The profession is far from unanimous as to its value. Even in Munich, a temple of praise for "606," where men bow their heads at its mys-

tic name, the joint administration of mercury and "606" is now being preached; doubt is apparently afflicting even the true believer. Does this heresy portend the passing of the cult?

The artlessness which named "606" far surpassed that which labelled beer XXX, or a soap powder 1776. "606" leaped at once to the level of a byword. Its notoriety spread as the plague until every layman in his club learned idly to trifle with the magic symbols. Because it was discovered in Germany, as a national duty many Germans lauded it and many Frenchmen did not. Claims as extravagant as its title were made for it; everybody rushed for it; and soon we were flooded with cures. Medical men not rarely found themselves in the position of the people of the kingdom in the fairy tale; there, you remember, two promoters had wonderful silk which could only be seen by those who were worthy. Here too many of us hastily admitted we saw the silk. And, now the reaction has begun, too many of us will as hastily deny its existence. I am not convinced that its effects are better than or as permanent as those we obtain from mercurial inunction; yet, except for the severity of the transient symptoms which so often follow, I have not seen untoward results in which the drug alone could be incriminated. It is speedy and efficacious, especially in exudative processes. But it is costly, inconvenient, and altogether too powerful a remedy to be lightly employed. The value of arsenic in syphilis has been known for ages. Donovan's solution, a combination of mercury and arsenic, has long been recognized as one of the most powerful antisyphilitic remedies in our pharmacopœia. Whether we can achieve the same results with massive doses of our ordinary preparations of arsenic as with this new compound, "606," I do not know. But with the cessation of popular clamor for the drug we shall be able to accumulate more reliable evidence and to determine precisely its true value.

Our next case, gentlemen, has had locomotor ataxia for many years, and has been bedridden for about eighteen months. His right third nerve is partially paralyzed; you see his right eyelid droops; he squints; and the movements of his eyeball inwards, upwards, and downwards are defective. He has double optic atrophy and nerve deafness. He has great difficulty in micturition; to begin the act is a serious task, and the urinary stream is degraded to a prolonged

trickle. Widespread sensory disturbances are present and affect mainly his deep sensibility. Yet, you observe, as I test him, that in addition to his loss of pressure pain he does not perceive pin pricks as pain.

The sensory impulses from the limbs and trunk pass to the spinal root-ganglia, through the peripheral nerves; thence they go through the posterior roots to the spinal cord. In lesions of the peripheral nerves we can distinguish usually between the area of loss to pain of pin prick and that of loss to pressure pain. An area of loss to the one form of painful stimulus may exist without any impairment of the other form, and when both forms are simultaneously affected in a peripheral nerve lesion the area of loss may vary for each. But in the spinal cord a rearrangement of fibres occurs; there is no more a separation into deep and superficial pain fibres; so that when disease involves the cord, as it does in this case, you observe that the perception of pain as a whole is abolished; the pain of pin prick and of pressure equally disappear.

Observe how he attempts to place his right heel on his left knee. The limb suddenly shoots up; waves wildly for a moment, bangs down upon the other leg, oscillates for a moment, and then zigzags up the tibia till it reaches the knee. If now he repeats the manoeuvre with his eyes closed, observe how he fails to reach the knee. He knows where he wants to place his foot, but unless he watches he has practically no idea of the actual position of his limbs from moment to moment.

In early infancy muscular acts are almost wholly reflex; the infant's grasp is merely a reflex response to a touch on its hand. By the aid of its vision the child acquires the power to coördinate, direct, and refine its movements. It consciously controls each act, but no sooner is an act acquired than it is degraded from a conscious to an automatic performance. Thus the first efforts to walk involve enormous mental strain; yet children soon walk without a thought of what they are doing. Some of us may remember our youthful days when we painfully learned to play the piano; how, looking intently from the music page to our fingers and from our fingers back to the page, we laboriously picked out each note. But after many weary days we could play without looking at our fingers at all. We had learned to dispense with our visual impressions and to rely solely

upon the deep sense stimuli which passed from our moving fingers, from the muscles and joints in action, along the peripheral nerves, through the spinal ganglia, into the posterior columns of the cord.

Now, in locomotor ataxia, as you know, the main stress of the disease falls upon the spinal ganglia and the posterior columns of the cord. The path of the deep sense impressions is destroyed; so the patient has lost the power of coördinated automatic action. Hence he is helpless and bedridden.

The next case, gentlemen, enters the room with his eyes intently looking towards the floor. The anarchy of his leg movements requires his constant attention. Observe how extravagantly he lifts his feet high in the air; his legs move in a contorted fashion which makes one marvel that he can walk at all. The downward movement of his feet is suddenly and unexpectedly arrested by the floor, so that at each step he seems to attempt to dig his heels into the floor. When I suddenly call "Halt," you see he sways and almost falls in his effort immediately to arrest his progress. It is as if his legs had suddenly surprised his body by stopping and inertia impelled the body still forward. Notice the labor and slowness with which he turns. And when he stands you see his feet are widely separated and his whole attitude is that of one performing a delicate feat of balancing. Yet he considers himself a second Blondin, for he was bedridden for two years before he was taught to walk again. I need not tell you that his pupils do not contract to light; that his deep reflexes are absent, and that he has the other classical signs of locomotor ataxia.

If his limbs are passively moved while his eyes are closed he is quite unconscious of the direction of the movement. The messages from his deep structures are almost wholly lacking. Yet he can walk. His gait, although inelegant and unconventional, is effective. What enables him to walk? You perhaps noticed how earnestly he watched every movement of his limbs; how in walking he looked constantly downwards at his feet. He is somewhat in the position of a child learning to stretch forth its hand towards a desired object; he is controlling his movements according to his visual impressions. And now if we make him close his eyes he at once tends to fall. When he stands his feet are widely separated so as to afford a broad base of support. And now when he begins to walk again move-



ment does not immediately follow his muscular effort; he must first tighten up his hypotonic muscles before they can exercise any force upon the loosely-jointed limbs; he increases his effort and the limb suddenly shoots forward till he consciously arrests its motion; and the floor violently stops his foot as he attempts to place it down again.

How are such patients taught to walk? How would one proceed to convert a depressed and hopeless bedridden patient such as the last into an eager, hopeful, happy fellow such as our agile friend here? No elaborate apparatus is needed; practically no expenditure is required. All that is necessary in most cases is merely intelligent interest on the part of the medical attendant. Impress your patients that they can and shall walk again. Encourage, persuade, and coerce them to persist during the first period of much striving and little accomplishment; your efforts will generally be successful. We usually begin the treatment by teaching the bedridden patient to control the lateral movements—adduction and abduction.

There is a small apparatus which any village carpenter can make and which you will find useful for this purpose. It consists of two oblong pieces of wood about one inch thick, nine inches broad, and as long as the breadth of the patient's bed. The one piece of wood is placed flat on the bed to act as a foot for the other, which is placed with one lateral edge along the foot-piece, so that the breadth of the wood is vertical and at right angles to the foot-piece. In the upper free lateral border of the vertical plank five semicircular notches are cut, sufficiently deep and wide easily to admit the heel. The whole of this upper edge should be carefully padded with cotton wool affixed by strips of adhesive plaster to prevent the patient injuring his ankles against the wood. This contrivance is placed at the foot of the bed and the patient is encouraged assiduously to practise lifting his foot from one notch to the adjacent notch without touching the intervening wood. The movement must be slowly and deliberately performed. The patient must intently watch every movement of his limb. As he improves he next acquires the art of moving his foot to alternate notches. At the same time the re-education of the hand may be begun. For this purpose we use a cribbage-board and pin, or a "solitaire" board and marbles, and train him deliberately to move the pin or the marble from one hole



or space to another. If the patient can sit up in a chair he may practise kicking a small ball placed near his feet.

The next procedure is to get the patient to stand. In the first half-hearted attempts to assume the erect posture he must be very carefully supported, for if he falls not only is he almost certain to sustain a fracture, but he is also apt permanently to be discouraged. As he gains confidence and power he may be encouraged to walk, supporting himself by means of a well-balanced, small, light arm-chair which moves on casters, and which he pushes in front of him; or, where practical, one of the many forms of walking machine—an adult size of the apparatus—in which babies are induced to take their first steps—may be employed.

When the patient can stand aided only by sticks, footmarks should be outlined on the floor by means of chalk. These footmarks should at first be close together and parallel, so as to involve only small steps which will not too greatly task the dawning balancing power. The feet should be moved alternately, each to its allotted footmark, with as much deliberation and accuracy as if a needle were being threaded. Soon one of the sticks may be discarded, later the other, and ultimately the happy consummation which you see in this patient is successfully attained. Don't let your patients practise too much, or they will become more ataxic and require a few days' rest. Their energies should be devoted to the quality and not to the amount of their exercise. There is no case that cannot be improved. And the degree of failure, gentlemen, is more often an index to the inadequacy of the physician than to the severity of the disease.

# Medicine

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## SPONTANEOUS GANGRENE

WITH THE RECORD OF A CASE ILLUSTRATING THE ORDINARY SENILE TYPE; RESULTING IN SPONTANEOUS AMPUTATION, AND A CASE OF SPONTANEOUS GANGRENE AFFECTING THE SEVERAL EXTREMITIES SUCCESSIVELY IN THE COURSE OF ACUTE PRIMARY INFECTIOUS ENDOCARDITIS

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## SENILE GANGRENE RESULTING IN SPONTANEOUS CURE

TEXT-BOOKS on surgery, as well as medical dictionaries, unite on defining gangrene as death of a part of the body in mass. While it may be due to destructive agents acting directly upon the tissues, it is also brought about by obstruction of the blood-supply. In one case it may follow a blocking up of the arteries; in another the venous return may be retarded; in another there may be stasis of the blood in the capillaries. Parts deprived of their nerve-supply frequently undergo gangrenous changes.

Senile gangrene is a dry form of the affection, and in most cases is due to obstruction of the arteries of the part, directly caused by disease of the vessels. In other words, there is present an obliterating endarteritis, or atheromatous changes in the vessel walls combined with feeble heart action. Ordinarily the obliteration of one or more vessels in a particular region is followed by no such sinister results, as the collateral circulation, the memorizing of which caused so much labor in our student days, soon restores the blood-supply. Here the vessels making up the collateral circulation are also diseased, or the affected artery is the sole source of supply. Embolism or thrombosis may also bring about this obstruction.

Consequently, the affection is most often encountered in the aged

and debilitated. It is seen with comparatively great frequency in homes, almshouses, etc., and seems to occur most often in men. The extremities, naturally, are the sites of the disease, the lower being more often involved. The toes and the heels are the usual locations, on account of the circulation being less vigorous here than elsewhere. When the obstruction to the circulation is gradual, the parts dry and mummify, but the same causes acting in a precipitous manner may cause death of the part before this mummification begins, thus producing moist gangrene.

As would naturally be supposed, the causes of senile or dry gangrene are various, and are those that have to do with the production of chronic vascular diseases and trophic disturbances generally.

A slight injury to the foot or to one of the toes is the beginning of the local condition in many cases. The ordinary black-and-blue discoloration which we associate with a bruise or contusion appears, but fails to disappear after a reasonable period, and is replaced by a deep purple hue. Surrounding this purple area, which continues to get darker in color, there is considerable congestion. Soon a boundary line appears, marked by a few blisters or a slight discharge of pus, beyond which the redness becomes more pronounced than formerly. The gangrenous portion now becomes black. This line is termed the *line of demarcation*, but does not form until the affection has reached its full extent, and represents Nature's effort to dispose of the dead part by producing inflammation in the intact portions. Sometimes this is of no avail and the condition advances; in many cases it is successful, or at least suggests to the surgeon how a successful result may be accomplished. Not infrequently this condition of gangrene will be attended with fever; this is more especially true when the suppurative process begins. As a rule, the constitutional disturbance incident to the gangrene is very slight. A very little pain or distress in the part attends the condition. Sometimes, when the condition is bilateral or symmetrical, it is a part of a condition known as Raynaud's disease. Again, it may follow the continued ingestion of ergot, such as has been observed, especially in certain European countries, as the result of contamination of rye grain with the ergot fungus. It may also be observed as a symptom of diabetes, or granular degeneration of the kidneys. Ashhurst refers to an investigator by the name of Tricomi who claimed to have

found a peculiar bacillus in the blood of patients suffering from senile gangrene.

The treatment of senile or dry gangrene presents but few alternatives. Those whose circulation has been shown to be poor should be given all the advantages that will keep it as near normal as possible. The internal administration of tonics is highly essential. Good food and rest are absolutely necessary. The urine should always be examined, as diabetes may exist in very slight forms unsuspected until a gangrenous spot appears. Hot applications, elevation, and gentle massage are indicated early in the gangrene; later, if the inflammation becomes pronounced, ice compresses should be substituted. W. Rapke<sup>1</sup> strongly advocates active hyperemia in the treatment of arteriosclerotic gangrene, and reports the case of a man in whom, after amputation of the toe, the condition continued to progress and was checked only by daily hot foot-baths, which restored pulsation in the vessels. Weak carbolic acid solutions (3 per cent.) relieve the inflammation and destroy the characteristic odor. It is customary to wait until the line of demarcation forms before resorting to surgical intervention, but often it will be necessary to postpone the operative treatment indefinitely on account of the patient's age and general enfeeblement.

In the case depicted in the illustrations, the Frontispiece presents every feature of a typical instance of senile or dry gangrene, with the additional unusual feature that the fingers and hand were attacked first, and Fig. 2 the still more unusual termination of the case.

The detailed history of this particular case reads as follows: Case 4397, Howard Hospital, S. K., aet. 63 years. Admitted November 24, 1910, discharged December 10, 1910. The patient was Jewish and a resident of Philadelphia. Upon admission his temperature was 98° F., but within a day rose to 100° F., declining to 97° F. within 12 hours, followed by a subsequent rise to 100 again within the next 12 hours. It again dropped to 98° F., and for the rest of his stay in the hospital it hovered around 98° F., the evening temperature being close to the normal, while the morning was 98° F. or a few fifths below. The pulse and respiratory rate were proportionate to the temperature for the most part. Urine was voided freely and the stools were normal. The nurse's notes record that the appetite was good and that he slept well. An ice-cap was placed on the head in the early part of the treatment, supplemented with wet compresses of 3 per cent. carbolic solution to the affected areas.

Urinary examinations were made almost daily, and were practically the same as regards results. The reaction was acid for the first five days and the last five days. It was alkaline and neutral in the intervening period as the

FIG. 2.



End-result of hand affected with dry gangrene. (For appearance of hand, with its gangrenous fingers, see colored frontispiece.)





result of medicines taken by the mouth. The specific gravity varied from 1007 to 1024. For the first two days there was a slight trace of albumin, but this disappeared entirely. Sugar was never present, and acetone and diacetic acid were impossible of demonstration. The microscopic examination showed a few hyaline casts, granular detritus, cylindroids, and triple phosphates.

The blood examination showed 4,210,000 reds and 20,880 whites, the latter dropping to 13,120 after the patient had been in the hospital twelve days. The blood-pressure taken in the right brachial artery was 126 systolic, 90 diastolic; in right forearm, systolic 116.

The patient, a married man, in apparently perfect health, was admitted to the hospital with pain in the fingers of the left hand and a frank senile gangrene of these parts. The family history was negative.

He was a teacher by occupation and was very regular in his habits, using alcoholic beverages, and tea and coffee in moderation, and tobacco not at all. His previous medical history was decidedly negative, never having had any diseases except those of childhood, but he stated he was very susceptible to coughs and colds.

Fourteen months previous to admission the patient first experienced a burning, tingling sensation in his fingers and toes. Soon the great toe of the right foot showed cyanosis and dryness of the skin, and several bluish-black spots appeared over the area of the right tibia; one of these ruptured and discharged pus. He was treated at the Jewish Hospital for nine weeks, during which the lesions disappeared, but he was unable to walk by reason of the pain and burning in his feet. This condition persisted until five months before admission, when the tips of the fingers of the left hand began to show the first signs of dry gangrene. In one week's time the process had spread to the first joint, but the succeeding progress was much slower. The pain was of an intense, constant, aching character, often so bad that the patient was unable to sleep. The man stated that even the mildest pressure over the posterior surface of the shoulder caused increased pain in the affected hand. There was no increased thirst nor frequent micturition, but the patient was constipated.

The physical examination was negative in its findings. The eyes were normal. The mouth was healthy, although most of the teeth were gone and those remaining were in none too good condition. There was a right inguinal hernia, although otherwise the abdomen was apparently normal. The heart and lungs were normal.

The left hand showed dry gangrene extending to the metacarpophalangeal joint, with pus about the line of demarcation. The left arm was pulseless up to the axilla, and there was only a faint pulsation in the axillary artery.

The patient was at this time seen by Dr. A. C. Wood, and we both strongly advised the necessity of an amputation of the forearm. Our advice, however, was positively refused, and the patient returned home.

Subsequently we learned that in the course of the next month the fingers dropped off, one by one, granulations were established, and three months afterwards the hand presented the very good appearance shown in Fig. 2.

Fortunately for our mortality statistics, few patients adopt the course detailed above, and it is probable that the rather unusually good general condition of our subject enabled him to prove a marked exception to the general rule.

Another unusual feature encountered is the occurrence of the condition in a part not usually regarded as dependent. While dependent parts are affected most frequently, we note that the condition is uncommon in paralytic limbs. Although the trophic foot-ulcers of locomotor ataxia may be regarded as gangrenous, they belong to the moist variety, and could not possibly be confused with senile gangrene.

The sex of the patient follows the customary rule; thus Mr. Pott long ago observed that, while each sex is liable to it, he had seen the condition much more frequently in males than in females. The age of the patient was somewhat less than usual, but no time of life is exempt when the blood-vessels are the seat of disease. Sir William Lawrence, in St. Bartholomew's Hospital, in London, 1857, observed a case in a young man 23 years of age, but this patient had recently had an attack of acute rheumatism followed by pericarditis and effusion.

From the absence of the other causes and from the age of the patient we must assume that in this case disease of the vessels had to do with the production of the gangrene. T. Holmes<sup>2</sup> states that as far back as the days of Cowper,<sup>3</sup> the ossification of the arteries was noticed in persons who had died of this disease.

This factor in the etiology of the disease was noted by many of the early writers on surgical subjects. No less an authority than Ambroise Paré expressed himself regarding the predisposing cause of gangrene to the effect that "the arteries are so shut or pent up in a strait that they cannot perform their motions of contraction and dilatation whereby their native heat is preserved and tempered. (English translation by Thomas Johnson, 1649.)

Coming down to more recent times we find these early observations corroborated by other surgeons, with perhaps a difference in phraseology. Thus Robert Liston,<sup>4</sup> *Elements of Surgery*, 1842-44, states that the most common cause of spontaneous gangrene of this character is a rigid state of the arteries in consequence of the deposition of calcareous matter between the internal and middle coat; this calcareous degeneration, he further observes, may be confined to a part of the limb or may pervade the whole of it.

J. M. Chelius<sup>5</sup> quotes Travers as having mentioned among the causes of gangrene such deep and extensive effusions as compress and

annihilate the internal circulation of the part. Thus he states, "I have seen a subfascial effusion following a severe strain of the forearm, producing a spreading gangrenous inflammation of the extremity to within a hand's breadth of the axilla; and similar cases of suppuration, between the deep-seated muscles of the thigh, I have known terminate suddenly in gangrenous inflammation of the entire limb to the groin."

It is well known that certain injuries and diseases of the nerves of a part may terminate in gangrene, but it is surprising to learn that the celebrated French surgeon, Larrey (quoted also by Chelius), believed that injury to the nerves in the course of ligation for popliteal aneurism was the cause of the gangrene that followed. It seems incredible that he should have entertained such a view in the presence of such obvious fact.

A review of the literature both remote and recent affords us no new information on the etiology, pathology, or treatment of this form of the affection and we must patiently await that day when arteriosclerosis and senility have been eliminated before we can entertain the hope that senile gangrene can be entirely prevented.

#### SPONTANEOUS GANGRENE AFFECTING THE SEVERAL EXTREMITIES SUCCESSIVELY IN THE COURSE OF ACUTE PRIMARY IN- FECTIOUS ENDOCARDITIS

The history and clinical course of the case presenting the features enumerated in the above title read as follows:

Case 3499, R. D., Italian, aet. 42 years, butcher by occupation; residence, Philadelphia; was admitted to the Howard Hospital, December 10, 1909, with a diagnosis of typhoid fever, which later proved to be a case of malignant endocarditis, in the course of which he developed gangrene of three of the extremities, and died April 10, 1910. Autopsy was performed by Dr. Norman Gwyn, pathologist to the hospital.

The temperature on admission was  $100\ 2/5^{\circ}$  F., the pulse 86, and the respiratory rate 24. Within a few days the temperature chart showed a decided septic condition, the morning temperature being close to  $99^{\circ}$  F., and the evening rise approximating  $101^{\circ}$  F., sometimes slightly higher. This continued for nineteen days, after which it rose to  $104^{\circ}$  F., dropping to normal at the end of 48 hours, this phenomenon occurring immediately after amputation of his left leg for the gangrenous complications. Within a few days the temperature assumed its septic character with less pronounced diurnal variations, seldom going above  $100^{\circ}$  F., often going to  $98\ 2/5^{\circ}$  F. and lower. On the 42d day of the disease it assumed a continuous type, hovering around  $100^{\circ}$  F., which it maintained for about 10 days, again returning to its original intermittent character. On the 68th day it became continuous, seldom going above  $99^{\circ}$  F.,

which characteristic lasted until the 83d day, when it dropped to 98° F. It continued to remain at 98° F. for the morning temperature, rising to 98.40° F. for the evening for 19 days. Then it began to rise, and within the next three days reached 104° F. At this time the patient had convulsions. The high temperature continued for about two weeks, terminating with an abrupt rise followed by gradual lowering until dissolution supervened.

Owing to the patient's inability to speak English and the absence of a dependable interpreter, the family history and past medical history were not obtained, and reliance had to be placed upon the physical condition entirely.

The physical examination made upon admission was negative in its results. There was some enlargement of the spleen and leucocytosis, but there were no frank heart murmurs. There were no chills, but the patient did complain of chilliness and there was more or less profuse perspiration. After a few days in the house, pain developed in the left thigh, accompanied by enlargement of the femoral artery, followed by its obliteration and subsequent gangrene of the foot and leg. At this time only unusual rapidity of the heart was noticeable.

Amputation of the gangrenous part was performed, and following this a basic murmur was developed over the heart, which later became apical in situation. Adventitious heart-sounds then continued for the rest of the course of the disease, but were inconstant in character.

On March 30 (the 111th day of the disease), a friction sound was heard in the third interspace to the left of the sternum. No apex impulse was visible. There was some increase in the transverse dullness at the apex. The inferior border of the spleen extended to a line with the umbilicus.

On April 1 (the 113th day), râles were heard in the right upper lobe, sibilant in character, a few subcrepitant. A systolic murmur was heard in the aortic area.

The urinalysis was negative from the time of admission until the 33d day of the disease, when red and white blood-cells made their appearance. Once or twice previous to this, however, a trace of albumin was noticed, but this was not constant. Within a few days these features disappeared entirely. On the 106th day albumin, red and white blood-cells, and granular and hyaline casts appeared and continued until the disease terminated.

The blood examination on admission showed hæmoglobin 90, red blood-cells 5,080,000, and white 12,800. Repeated examination showed but slight variations. Once the hæmoglobin was 58, the red blood-cells 4,920,000, and the white blood-cells 28,480. Blood cultures were made, but were negative in result. Vidal's test was taken several times and was likewise negative. The sputum was examined several times with negative result.

Four days after admission, patient complained of pain in his left leg, followed by chill and perspiration. He continued to have pains in same limb, the foot of which became bluish in color on the following day. The pain continued and the condition of the foot progressed to gangrene, so that it was deemed best to amputate it on the 18th day after admission.

Two days later he complained of pain on his right side. On the 28th day he complained of pain in the right foot and leg. This continued, and one week later the right foot began to feel cold and numb, and within 24 hours it became mottled in appearance. It became progressively darker in color, and the severity of the pain increased. Gangrene shortly supervened, and amputation was performed on the 52d day.



Later pain was experienced in the right thigh, and it was necessary to amputate this member also. On the 83d day of the disease, after having been up and about his room for a few hours daily for several days, he developed convulsions. This was the beginning of the albumin and casts in the urine. Convulsions appeared later from time to time. Following this the tips of the fingers began to feel cold, and gangrene of the right hand and arm was soon observed. Blood-streaked expectoration and pains over the heart were more or less constant during the last ten days or two weeks of the disease.

At autopsy ulcerative endocarditis, especially of the mitral valve, was found, but there were no other gross pathologic lesions except as noted. The details of the autopsy as performed by Dr. Norman Gwyn read as follows:

*Chest Wall.*—Erosion of chest wall.

*Heart.*—Left chamber, large ball thrombus, 2 Cm. x 1 Cm., firmly attached to columnæ carnæ, evidently of some duration. On posterior flap of mitral valve, above edge of contact there were several old warty vegetations. In right heart, on the posterior leaf of tricuspid valve there was a large deposit of fresh warty endocarditis. Small atheromatous patches of aorta were observed. Heart-muscle was of a good color.

*Lung.*—*Right.*—Purulent pleurisy and gangrene of upper and of lower lobe present. Apex and diaphragm adherent and broken into two purulent collections, one along junction of middle and lower lobe in posterior capillary line, the other at the base behind these, leaving two ragged openings which have numerous tracts into the lung; smaller abscess cavities on section. No fresh infarcts are seen; major portion of lung tissue collapsed and airless. *Left.*—Shows no gross infarcts, one depressed ulcerated tear on apex posterior lobe, small abscess toward base, size of a bean, several the size of peas. Bronchopneumonic condition at apex, marked excess of fluid, no consolidation.

*Diaphragm* is raised on right side in 4th interspace, on the left in 5th interspace, and is adherent to an erosion coming from the interior of the chest. Clear fluid in left pleural cavity, also in right.

*Pericardium* contains several C.c. of clear fluid, no distinct evidence of pericarditis.

*Suprarenals.*—Negative.

*Kidneys.*—*Left.*—Large and soft, artery and vein clear. Several scars in capsules slightly adherent. Cortex narrowed, marked cloudy swelling and congestion, one infarct in under surface toward top. None of the infarcts is fresh or soft. *Right.*—Artery and vein free, smaller than the left. Capsule adherent, numerous scars of infarcts, purple color. Much less evidence of parenchymatous change than in the left, scars are evidently old.

*Pancreas* is large and firm, and cuts with resistance at head, small necrotic area towards tail, where it embraced the spleen; duct seems clear.

*Rectum* shows several small ulcerations, marked congestion. *Appendix* normal, lying free. Small intestine shows small areas of congestion.

*Stomach.*—Enlarged, mucus in excess, otherwise negative.

*Liver.*—Large, pale, surface greasy. Gall-bladder retracted and contracted, some cloudy swelling and some increase of intralobular fibrous tissue.

*Aorta* clear. Left iliac artery is free and contains natural-looking clots, veins contracted. Iliac and caval veins filled with firm, adherent thrombi. Carotid, brachial, and subclavian arteries in the right side are free.

*Spleen.*—The peritoneum was adherent and there were two healed, wedge-

shaped infarcts made up of partly-organized firm material of a yellowish-white color.

In analyzing the case we find it is most unusual in many particulars. Spontaneous gangrene from any cause cannot be regarded as common, and spontaneous gangrene attacking the several extremities in succession must be regarded as decidedly uncommon, while the affection, being shown to be directly due to valvular heart-disease, is sufficiently unusual to entitle it to more than passing consideration.\*

In perusing the literature on the subject, one of the earliest cases of gangrene of both extremities we find recorded by Ambroise Paré (English edition by Thomas Johnson, 1649), who refers to "A certain man in Paris, who, supping merrily and without any sense of pain, went to bed, and suddenly in the night-time a gangrene seized on both his legs, caused a mortification without tumor, without any inflammation; only his legs were in some places spread over with livid black and green spots, the rest of the substance retaining its native colour; yet the sense of these parts was quite dead, they felt cold to the touch; and if you thrust your lancet into the skin no blood came forth." The patient died despite treatment. In this case, from the sudden onset it must be assumed that embolism was the immediate cause, while the most common underlying cause of the embolism must have been valvular heart-disease. This is entirely speculative, as there was no autopsy recorded.

Thomas Annandale<sup>6</sup> records several cases in which spontaneous gangrene occurred incidental to obstruction within the main artery of the affected parts. He refers to the case mentioned by John W. Ogle<sup>7</sup> in which gangrene followed an arrest of circulation in the main arterial branches of both arms and legs.

J. M. Chelius<sup>8</sup> mentions a case following scarlet fever, another following simple continued fever, one during the course of typhus fever, and still another in a man forty years of age who had organic heart-disease. It is not difficult in these particular febrile cases to read into the account a septic condition of the cardiac valves.

Travers, on the other hand, was unable to associate many of his cases with arterial disease, believing them to be idiopathic in origin in many instances, but Brodie saw a fatal case, in a man, which he attributed to extensive inflammation of the principal vessels of the part.

Solly<sup>9</sup> records the case of a boy, three years old, in which the gangrene spreading from limb to limb destroyed the boy when he was four years old. It began in the left foot and involved the leg; then it attacked the right foot and leg. Next the right hand and arm; then the left hand and arm, and lastly the tip of the nose was involved. The age of the patient eliminates the ordinary arteriosclerotic processes from the etiology. The history and absence of contagion do not place it in the class of "hospital gangrene" of the older writers, so we must assume that embolism was the immediate cause of the condition.

J. Spence<sup>10</sup> records a fatal case of spontaneous gangrene of the lower extremity in which he demonstrated, by autopsy, atheromatous and calcareous

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\* Report of three cases of malignant endocarditis, one following measles, another typhoid fever in a child and simulating splenic lymphatic leukæmia, and another terminating in recovery, by Albert Roussel, M.D., *The Medical Record*, April 20, 1901.

degeneration of the vessels of the thigh. It is not likely that this was the first case in which an autopsy was performed with such results, but, as it is one of the earliest in the literature available, it is mentioned here.

J. R. Begg<sup>11</sup> reports a case of idiopathic gangrene of the four extremities and the nose and ears, in which the patient recovered after amputation of the extremities. The case occurred in the person of a young married woman about twenty-one years of age. She had just been confined and been delivered of one child. While rather weak following labor she suffered no complications or after-effects. During the progress of her parturition she had been given 65 grains of ergot in infusion in two doses. About two weeks after labor she noticed blueness of the nose, left hand, and left foot, and later the right hand and right foot were involved, followed later by blueness of the lobules of both ears. Both she and her husband stated that her extremities had always shown deficient circulation. The parts mentioned soon became the site of dry gangrene. This began about 46 days after the accouchement, and 40 days after the commencement of the gangrene both legs were amputated. Later the tip of the nose and the lobules of the ears were removed; 22 days after the amputation of the legs both arms were amputated. Recovery was prompt. The entire affection from the beginning to absolute recovery occupied four and a half months. Some feature of the case suggested ergotism, but Beggs did not think this was the cause in this case, especially as it occurred as a single isolated case and the dose of drug used after labor was comparatively small. There were no gross evidences of arterial or venous obstruction, heart-disease or other condition to attribute the gangrene to, so that Beggs assumed it to be purely idiopathic in nature.

The curious feature of this case is that Beggs should regard it as idiopathic in character. It seems reasonable to suppose that clots from the site of the placenta may have had something to do with the condition.

M. L. Stevens,<sup>12</sup> Asheville, N. C., contributes the history of a fatal case observed by him occurring in a young girl thirteen years old, in which the left foot and leg and the right foot and ankle were attacked. There was clinical evidence of endarteritis and thrombosis of the nutrient arteries, but no evidence of heart-disease. As there was no autopsy, it was difficult to say what other gross conditions were present.

Thurston<sup>13</sup> refers to two cases of spontaneous gangrene which he observed in India. In one case the patient was a man fifty years of age, and the arm was the part affected. In the other the patient was forty years old, and the leg was the part involved. He regards these cases as due to embolism and thrombosis, possibly secondary to a weak heart.

H. J. Lee<sup>14</sup> gives an account of a case in which a woman was the subject of an uncomplicated case of pneumonia; the crisis occurred on the eighth day, during which the patient was very weak and acute dilatation of the heart developed. About ten days later she experienced pain in the knees, and still later the toes and feet became cold. Subsequently gangrene followed in both legs and amputation was necessary. Septic pneumonia supervened, but the patient eventually recovered.

In cases of symmetrical gangrene the liability of confusion with Raynaud's disease is very great, but even in the latter some observers ascribe an infectious character to the condition. In passing, mention may be made of the eight cases

of symmetrical gangrene reported by Edward Jenner Wood,<sup>15</sup> of Wilmington, N. C., which he attributed to malarial infection. His paper is rich in bibliographic data concerning all the recorded cases of a like character.

Infantile gangrene is a very uncommon condition, yet Sassi<sup>16</sup> observed two fatal cases affecting the feet in breast-fed infants, both of whom had chronic enteritis. Vascular disease was believed to be the cause of the gangrene.

Another case occurring in an infant provides the subject of a paper by Beek.<sup>17</sup> According to the account thus given, the patient was the first-born of healthy parents, and was apparently healthy until nearly five months old, when the mother noticed recurring capillary congestion and bluish discoloration of the hands, feet, and ears. In the course of six weeks the cyanosis recurred daily and gangrene developed. The child died within a month with signs of heart weakness, and atrophy and gangrene of the left hand. The autopsy showed patches of thickening of the intima of all the arteries of the extremities and some of the veins of the arms. Otherwise the findings were negative.

Spinal anæsthesia was the cause of the condition in a case recorded by Sudeek.<sup>18</sup> The patient was a woman, sixty-two years of age, and had been anæsthetized by the spinal method for an operation for the relief of an incarcerated hernia. The following morning redness and vesication were observed on the dorsum of the feet and part of the toes, which ultimately became gangrene. He refers to a similar case observed by Goldman in which the heels were affected.

R. Dexter and A. W. M. Ellis,<sup>19</sup> of Cleveland, Ohio, reported a case of spontaneous gangrene observed by them which they believed was due to general arterial thrombosis. Their report is most valuable by reason of the exhaustive review of the literature which it contains. They found 73 recorded cases which they considered undoubted examples of gangrene, 69 of which occurred in males and 4 in females. Two patients were under twenty years old; 7 were between twenty and thirty; 9, between thirty-one and thirty-five; 22, between thirty-six and forty; 12, between forty-one and forty-five; 10, between forty-six and fifty; 10, between fifty and sixty; and 1 in which the age was not mentioned. Although the symptoms in one case were present for over 20 years, the average duration was from 3 to 5 years. In the 68 cases in which the location was mentioned the condition occurred 53 times in the legs alone; twice in the arms alone, and 13 times in both upper and lower extremities. In more than one-third of the cases the pulse in the femoral and all the vessels distal to it was lessened or obliterated, while in nearly two-thirds the vessels as high as the popliteal were involved. Neither syphilis nor the use of tobacco was a factor in the etiology.

F. J. Grant<sup>20</sup> describes in detail a case of spontaneous gangrene in a boy, sixteen years of age, attended with heart-disease. The onset of the gangrene was sudden and its course rapid. At the beginning the physical signs regarding the heart were absolutely negative, with the exception of feeble heart action. There was a diffused forcible pulsation of the external jugular vein. There were also emphysema and slight bronchitis at the base of both lungs. Occasionally, as the disease progressed, and especially after separation of the sphacelus, the heart action became very feeble and its impulse scarcely perceptible. Violent attacks of dyspnoea, threatening instant suffocation, occurred at intervals, and, in conjunction with these, failures of the circulation. Epilepti-



form convulsions were observed as the condition advanced. There was slight albuminuria, but no glycosuria. The diseased portions were removed when the line of demarcation was defined. From time to time symptoms of venous circulation with dyspnoea and cerebral disturbance or obscurity and unconsciousness were observed. Death eventually supervened.

From the results obtained at the postmortem examination it would appear that in Grant's case the gangrene of both feet, depending evidently on systemic venous congestion, was caused by the enlarged state of the auriculoventricular openings and the accompanying dilatation of the auricles, and of the right ventricle in a lesser degree. It would also appear that the gangrene was essentially cardiac in origin, although the cause of this might have been exposure to cold. The immediate cause of death was found to be pericarditis, pleurisy, and capillary bronchitis.

Cabot and Warren<sup>21</sup> give the history of a case of acute spontaneous gangrene, occurring in a man twenty-five years of age, attacking the right leg. The patient died 10 weeks after the beginning of the disease, and autopsy revealed thrombosis and complete closure of the right external iliac artery. There were metastatic abscesses of the kidneys, spleen, liver, and lungs. These authors believed that the thrombosis was dependent upon syphilitic endarteritis.

Pneumonia at times is a factor in the causation of gangrene. McGregor<sup>22</sup> gives an account of a case of gangrene of the fingers of the right hand following pneumonia. He believes the condition may be due to the pneumococcus acting directly on the vessels producing thrombosis, or to an endocarditis (pneumococci) giving rise to embolism and thrombosis, or both more or less in combination.

Typhoid fever also has an important place in the etiology of some cases of gangrene. Birow<sup>23</sup> found eleven cases in Russian literature of gangrene of the extremities complicating typhoid fever, and adds the record of one he personally observed. With but two exceptions, the patients were between eighteen and twenty-five years of age. In his case, gangrene of the right foot developed at the beginning of the third week of the typhoid.

While the usual practice in the treatment of gangrene is largely expectant until the line of demarcation is defined, occasionally some novel treatment is instituted that is attended with success. Thus Wieting<sup>24</sup> records an instance in which he accomplished a successful transplantation of blood-vessels in a man of forty years of age with angiosclerosis of the vessels of the legs. The right leg had been amputated for gangrene two years previously. The left leg became involved two months prior to the operation upon the blood-vessels, which consisted in implanting the femoral artery into the femoral vein just below the mouth of the saphenous vein. The operation was performed under spinal anaesthesia.

Gangrene of the foot has been known to follow influenzal infection. This is evidenced by the record of a case reported by C. W. Allen.<sup>25</sup>

Pregnancy has been attended by spontaneous gangrene. R. Jardine<sup>26</sup> contributes to the literature of this subject the clinical history of a case of gangrene of the leg from thrombosis, occurring during pregnancy.

H. W. Fuller<sup>27</sup> gives an account of a case of spontaneous gangrene in a woman thirty-seven years of age, in which the condition first attacked the right foot, leg, and thigh, and then appeared in the left foot, involving successively



the leg and thigh. Death supervened shortly, but postmortem examination failed to reveal any disease of the heart or its valves. The lungs and spleen were congested and the right kidney was the subject of granular degeneration. The great blood-vessels of the lower abdomen were filled with firm coagula.

J. Cockle<sup>28</sup> relates the course of a case of arrested circulation in the lower extremity presenting the premonitory symptoms of gangrene occurring in a woman forty-one years of age. When first seen the patient was apparently suffering from acute rheumatism. Physical examination revealed embarrassment of the heart action, and a loud murmur audible in the precordial region, particularly over the origin and course of the aorta. The patient died four days after the first observations were made. Autopsy showed enlarged heart with dilated left ventricle. The mitral valve was thickened. The leaflets of the aortic valve were thickened and incapable of accurate closure of the opening. No vegetations on any of the valves. The ascending aorta was dilated and considerably diseased. The liver was enlarged and the stomach and spleen were displaced. The femoral arteries on both sides were diseased and contained clots. Cockle regarded the gangrenous condition in the extremities as due to the lodgment of emboli in the large vessels, secondary to the heart disease. He refers to two cases of sudden arrest of the circulation in the brachial artery, the records of which are to be found in the first and third volumes of Lentin's *Beiträge*, under the headings "Gangrenous Spontaneus" and "*Herzlähmung der Armpulsader*."

W. Caley<sup>29</sup> observed a case of gangrene of the lower extremities in a woman fifty-two years of age associated with mitral stenosis, thrombosis of the left auricle, embolism of the left inferior parietal lobule, and thrombosis of the femoral artery. The beginning of this series of disturbances was marked by sudden loss of consciousness, which lasted three hours. Following this, speech was lost and mental faculties impaired, but there was no paralysis. Gangrene of the legs supervened and death occurred on the sixteenth day of the disease, following an attack of facial erysipelas. Autopsy showed two areas of softening in the left inferior parietal lobule. The heart was enlarged, the left auricle dilated and hypertrophied. The mitral valve was thickened and calcified, and the orifice much constricted. The lungs were pigmented and emphysematous. The right kidney was granular. The femoral arteries were occupied by firm, adherent coagula.

Carter<sup>30</sup> contributes to the literature of spontaneous gangrene the clinical history of a case under his observation occurring in the person of a male laborer, forty years of age. For two years previously the patient had been the subject of dyspnea, cough, and edema of the feet after the slightest exertion. The heart was considerably enlarged, and a systolic bruit could be demonstrated at the apex and base of the heart. There was diminished sensation in the left arm and leg, and absence of pulse from the right wrist. The right forearm was intensely white and cold. The leg became frankly gangrenous, and the patient died after an illness of twenty-seven days. The autopsy showed that the right internal carotid and middle meningeal arteries were blocked by a firm plug, and the portion of the brain supplied by the latter was very much softened. Mitral and aortic disease were present, as evidenced by calcareous deposit on the valves. The right brachial and right common iliac arteries were blocked by coagula, and there were several infarcts in the kidneys. This is by far the most extensive case in available literature.

Spontaneous gangrene has been observed in the course of extreme anæmia, as is well illustrated in the case observed by M. L. Nash.<sup>31</sup> The patient was a very anæmic individual of from thirty to thirty-five years of age, and gangrene developed in both feet. Spontaneous amputation followed with complete recovery.

In some cases where the condition ends fatally the immediate cause of death is very doubtful. Thus in the case reported by R. Hills,<sup>32</sup> death resulted with symptoms of true or simulated hydrophobia. The patient was a married woman, forty-one years of age, and gave a history of having been bitten by a pet dog, while it was in some kind of "fits," when she was sixteen years of age. Her illness covered a period of 45 days, during which the right leg became gangrenous, requiring amputation. Convulsions developed shortly and the patient succumbed, with symptoms not unlike those of hydrophobia.

One of the older surgeons, Mr. B. Cooper, of Guy's Hospital, London,<sup>33</sup> once observed a case of dry gangrene of both feet in the case of a woman eighty years of age, which terminated fatally, and was shown upon autopsy to be due to ossification of the greater part of the large arteries, especially the descending aorta and the iliac arteries.

Mr. Travers<sup>34</sup> had the opportunity of observing a case of spontaneous gangrene of the leg in a man sixty-one years of age, with a fatal termination. The onset was sudden and the patient was first attacked with a severe pain in the stomach while at work. Shortly afterward gangrenous symptoms appeared. Autopsy showed dilatation of the left ventricle of the heart with hypertrophy, and disease of the mitral valve. The vessels of the affected part were not found to be the seat of arteriosclerosis.

Graves and Stokes,<sup>35</sup> in a dissertation on diseases of the arterial system, relate the history of a case of arteritis in a man, forty-four years of age, affecting the right leg, causing loss of power and a reduction in its temperature. The femoral artery was hard and painful, and there was an absence of pulsation in the right common external iliac artery. Death supervened shortly, and, had it not, gangrene would certainly have followed, since autopsy showed the presence of a clot in the right common iliac artery.

From the Hospital des Enfants Malades<sup>36</sup> we take the record of a case of spontaneous gangrene occurring in a weakly child, three years old, in which the condition began with convulsions. Ten days later gangrene began, and three weeks after the beginning of the disease amputation of the right leg was performed. Gangrene then began in the left leg, and the patient died two weeks later. Autopsy showed the heart and aorta to be normal, but the lower part of the abdominal aorta, and the iliac arteries and their branches, contained clots. There were multiple abscesses in the spleen.

The newborn are liable to gangrene as well as those advanced in life. Kosmak<sup>37</sup> describes one case and refers to four others in medical literature in which gangrene occurred spontaneously in the newborn. He believes the condition to be a form of sepsis.

From the foregoing it would appear that spontaneous gangrene could occur only from obstruction of the main arterial supply of the part, this obstruction being due either to localized disease of the vessel walls, the swelling of which produces the obstruction, or to the series of phenomena that followed the lodgment of an embolus, the embolus usually being derived from diseased heart valves.

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- <sup>33</sup> *Lancet*, London, 1826, 224, *Hospital Reports*.
- <sup>34</sup> *Ibid.*, 1827, 731, *St. Thomas Hospital Reports*.
- <sup>35</sup> *Dublin Hospital Reports*, 1830, v, 631.
- <sup>36</sup> *Lancet*, London, 1829, 655, quoted from *Jour. Hebdomadaire*.
- <sup>37</sup> *Archives of Pediatrics*, 1908, July.

# THE DIFFICULTIES ENCOUNTERED IN DIFFERENTIATING BETWEEN ULCER OF THE DUODENUM, AND DISEASES OF THE CÆCUM, APPENDIX, AND LOWER ILEUM

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SINCE the invasion of the stomach by the surgeon, many hitherto obscure pathological conditions have been cleared up. At the same time we have learned to appreciate that diseases in closely allied organs, such as the gall-bladder, appendix, lower ileum and cæcum, may all present somewhat similar symptoms, so that frequently the very best diagnosticians fall into error.

Until the Mayos pointed out the dividing line between the stomach and the duodenum, we heard very little of duodenal ulcer. Since then, however, gastric ulceration seems to have taken a back seat, and we have gone to the other extreme. This is all very natural, because, notwithstanding the fact that abdominal surgery has made wonderful strides within the last ten years, we are still ignorant of the paths travelled by reflected pain, and of the vago-sympathetic relationship between these different organs.

Moynihan, in his work on Duodenal Ulcer, says: "There are few diseases whose symptoms appear in such a definite and well-ordered sequence as is observed in duodenal ulcer. It is true that there are cases, of which fuller details must presently be given, in which the regular appearance of the symptoms is absent, or in which one symptom is so exaggerated as to dwarf, or even to destroy, the value of others. But these exceptions are few, and they do not belittle the value of the general statement that the symptoms of duodenal ulcer are definite, and not easily to be mistaken, and that they appear in an order and with a precision which are indeed remarkable." Since then he has had reason to change his mind, and in an article which appeared in the *Lancet*, for January, 1912, he admits that the diagnosis of duodenal ulcer is by no means easy. In this article he is

inclined to believe that the infection that results from an inflamed appendix is very often the starting point of this ulcer, and he is now careful, no matter on which organ he operates, to examine the appendix before closing the abdomen.

A classical history of duodenal ulcer, according to Moynihan, is as follows: Weight, oppression or distention in the epigastrium after meals. Two to six hours after meals, pain comes on gradually, and gradually increases, accompanied by fulness and distention, and eructations of bitter fluid or gas. These eructations afford some relief. As a rule, in the beginning, the pain is noticed only after a heavy meal, but later, after almost every meal; it is relieved by taking food, or saline cathartics, or even a drink of water. Characteristic of the pain is that it wakes the patients up in the middle of the night, and they discover from experience that it can be eased by lying on the stomach on a pillow, or leaning over the back of a chair. They also learn that food affords some relief, so that most of them keep a little milk, or water, or crackers constantly at the bedside, or in their pockets. The pain is often accompanied by a sensation of weight, fulness and distention in the epigastrium, described as "boring," "gnawing," or "burning." It may be relieved by belching. Sometimes there is a slight regurgitation of food, which leaves a bitter or acid taste in the mouth, and the throat feels hot and scaldy, and again there is a gush of saliva, the swallowing of which affords some relief. At times the pains are cramp-like and colicky in character; Moynihan attributes this to the spasm of the pylorus. The appetite as a general rule is good, but on account of the distressing symptoms which follow the ingestion of food, patients dread eating and reduce their diet to almost nothing. Vomiting may, or may not, be present. In some cases the attack is precipitated by excessive smoking, or by severe spells of cold weather, and the "hunger" pain is especially so. According to Moynihan hyperacidity and hyperchlorhydria are rather characteristic. There is usually some tenderness in the epigastric region, generally in the middle line, especially to the right. Hemorrhages from the bowel occur only in the late stage. We mention this purposely, as we wish to show, later on, that disease in other organs, such as the appendix, lower ileum and cæcum, give the same train of symptoms, and that as time goes by we may have to add other organs to this list.



G. C., age 37, single. There is nothing in his history of any importance. He has never had any sickness, so far as he knows, and has always taken pretty good care of himself, taking a great deal of exercise, having been an athlete all his life.

Habits: Alcohol regularly; sometimes to excess. Smokes a great deal.

About two years ago he had an attack of indigestion, followed by nausea and vomiting. He attributed this to something he had eaten, and thought nothing more about it. A few days later he had a similar attack, with bloating and eructations, and severe pain in the epigastric region about three or four hours after dinner. Since then he has had more or less trouble, and within the last six months has been almost incapacitated from the pain and discomfort, so much so that he has lived on a diet of crackers and milk. The pain wakes him up in the middle of the night; but he gets some comfort by lying on his abdomen, on a pillow; it is eased by taking some food or water, but a Seidlitz powder gives him more relief than anything else. Consequently he keeps one in his pocket all the time. Occasionally he empties his stomach by tickling the fauces with his finger. Sometimes he has severe pain during the day, and by leaning on his abdomen over the back of a chair he has found that he gets some relief. Occasionally he vomits, and feels better afterwards. Sometimes the discomfort is so great, especially after some indiscretion, as excessive smoking, or drinking, that he has to walk the floor all night.

This patient being a personal friend, the author had occasion to observe him very often, and he knows that the man suffered the most excruciating agony. Physical examination was negative, except for some tenderness in the epigastric region. The man was exceedingly muscular, and had a good layer of fat, so that abdominal palpation was not by any means easy. There was no tenderness of the appendix, except what one might obtain by deep palpation of any part of the abdomen. He was put on a test diet, followed by an examination of the stools; given a test breakfast, and the stomach contents examined; the twenty-four-hour specimen of urine was also examined and a nitrogen and sulphate partition made.

This case was seen by Dr. Joseph Blake, in consultation with the author, and he agreed with the latter's diagnosis of duodenal ulcer. Dr. Blake consented to admit him to the Presbyterian Hospital, but the patient was suddenly called back to England, and had to leave on a few hours' notice. Shortly after his arrival in London he had a severe attack of tonsillitis, which laid him up for several weeks; following that he had an acute exacerbation of his old trouble, and was operated on by an eminent London surgeon. The operation revealed a gangrenous appendix, and careful examination of the stomach and duodenum failed to show any lesion. This case gave a typi-

cal history of duodenal ulcer, and confirmed the diagnosis made by the author after repeated and very careful examinations, and due deliberation.

On the other hand, we quote a case which has already been published, in the *New York Medical Journal*, where all the symptoms pointed to a chronic appendicitis, when a hemorrhage revealed an old standing duodenal ulcer.

CASE.—Mrs. M., age 30; no children.

Family history: Father died at sixty-five, of apoplexy; mother living, healthy. Brothers and sisters all healthy.

Habits: Patient drank tea, coffee and alcoholic liquors in moderation. Smoked six cigarettes daily.

Diseases of childhood: Measles and scarlet fever; no complications.

History: Patient had been perfectly well up to five years ago, when she had what was evidently an acute inflammatory process of the uterine adnexa. Since then she has suffered, more or less, from constipation. The present trouble dated back to February, 1909; following a rather constipated movement, she had pain in the anus, so severe that morphine was given to relieve it; she was fairly comfortable until the next movement, when she began to have severe pain radiating to the legs and to the back; continued in this condition for several weeks, having pain every time the bowels moved, but was made fairly comfortable by means of suppositories and laxatives. Constipation then became worse, and owing to the pain that accompanied it, she went to the toilet only once in two days. In May, 1909, she was referred to me.

She was rather stout, but anæmic. Examination of the heart and lungs was negative. Abdominal examination revealed nothing unusual, except some tenderness over the appendix.

*Vaginal Examination.*—Uterus retroverted and retroflexed. A good deal of thickening over the left tube, which was bound with a mass of adhesions to the sigmoid. The right ovary was prolapsed, enlarged, and fixed in the posterior cul-de-sac.

*Rectal Examination.*—The sphincter spasmodically contracted. There was an anterior sentinel pile, above which could be seen a well-developed fissure. Examination was so painful that no further effort was made to explore the rectum at this time. Local applications to the fissure gave the patient great relief and in a short time it was entirely healed. A proctoscopic examination revealed atrophic, catarrhal condition of the rectum and lower portion of the sigmoid. It was impossible to pass the proctoscope above this on account of an acute angulation of the sigmoid, evidently due to an adhesion to the left tube. Three months afterward I was called to see this patient. There was a recurrence of the fissure, severe pain over the appendix, exquisite tenderness on pressure, and all the classical symptoms of appendicitis. A diagnosis of acute appendicitis was made and operation advised. That evening she passed a lot of dark clotted blood and fecal matter; subsequently she had a number of movements, dark in color, and containing large quantities of digested blood. An examination of the rectum under nitrous oxide gas revealed nothing except what

has already been stated. Next morning the patient was given a Boas-Ewald test breakfast.

*Macroscopical*.—Large quantity of liquid with very little solids.

*Microscopical*.—Starch, yeast, and a few epithelial cells with fat globules.

*Chemical*.—Free hydrochloric acid, 70; combined acids, 24; total acidity, 90; lactic acid, a trace; starch digestion, poor.

Here was a patient who, until she was cross-examined, gave no history of any disturbance of digestion. Her pain was all referred to the region of the appendix, and the tenderness was confined to the same region. Later on I elicited the fact that she suffered off and on from what she called indigestion; that she occasionally had pains coming on some four or five hours after eating; took something for relief, vomited, and felt better.

Since the appearance of Jackson's article, we have learned that these adhesions play an important part in causing reflex symptoms, which closely simulate ulcer of the stomach.

Sir William McEwan, many years ago, pointed out the influence of the appendix on secretion and digestion, and was able to show that when the appendix was stimulated the secretion increased, but if this stimulation was kept up for a long time, the secretions gradually diminished, and finally disappeared. This is borne out clinically by the fact that most cases of acute appendicitis are ushered in with an attack of diarrhœa, and that chronic appendicitis is nearly always associated with constipation, hyperacidity of the stomach, and spasm of the pylorus.

The following cases will illustrate both points:

CASE I.—S. M., age 35; married.

Family history: Father living, 80 years old. Mother died of typhoid fever.

Habits: Alcoholic liquor in moderation, four cups of coffee daily, and about fourteen cigarettes.

History: Gonorrhœa twenty years ago, otherwise perfectly healthy up to one year ago. He suffered from piles and bleeding every time his bowels moved, but couldn't say positively how much blood he lost, as he only noticed the blood on the paper. During and following a movement he suffered severe burning pain, which would last for an hour or two; he had three or four movements daily. This state of affairs (that is, burning pains, blood in stools, etc.) lasted for six months, when the pain and burning disappeared. He has suffered for the past six months from a pain in the stomach, coming on about two hours after eating, with belching gas, and acid eructations, and a burning sensation. The distress is relieved by taking food.

The principal pain was located between the umbilicus and the ribs, radiating

to the right side. He very often woke up at about three o'clock in the morning with severe pain, which was somewhat relieved by lying on the stomach. He suffered from headaches, a tired feeling all the time, especially after eating, and was exceedingly nervous.

The patient is a rather thin individual, with a muddy complexion, very nervous, amounting almost to hysteria. His arteries are thickened, pulse full and bounding, blood-pressure high,—190. Lungs, negative. Hypertrophic dilatation of the heart. His chest is long and narrow. A narrow costal angle is the other evidence of the *habitus entropicus*, Lennhoff's index. There is a partial separation of the rectal muscles. The abdomen is of the scaphoid type, the stomach in normal position. The cæcum is enormously dilated and thickened, and filled with fluid, and the outer border rolls up over the finger. Palpations over the cæcum cause severe pain, radiating first to the umbilicus and then into the pit of the stomach, similar to the distress after eating; some pain is also felt in the small of the back. The patient refused to have a proctoscopic examination. He was given a Boas-Ewald test breakfast; this was removed three-quarters of an hour afterwards. The total acidity was 96. No blood.

Diagnosis: Mobile and dilated cæcum, with a probable Jackson's veil.

He was given  $\frac{1}{4}$  gr. morphine four nights later, on entering the hospital, and being a very nervous patient, he was given another  $\frac{1}{4}$  gr. the next day, and  $\frac{1}{8}$  gr. before the operation.

As usual the skin was bathed with tincture of iodine. Battle incision was made about two inches long. On opening the peritoneum, the cæcum appeared, and was covered by a warm saline pad. The cæcum had a long mesentery, similar to that described by Professor Wilms, of Heidelberg (mobile cæcum). Examination showed the cæcum covered by cobweb adhesions (Jackson's), extending to the appendix. (Fig. 1.) The latter was pulled down in the middle by strong adhesions, which formed an acute angle. The scissors were passed underneath the cobweb adhesions, and by this means these were separated from the peritoneum. Using scissors for a director, a double catgut ligature was passed between the scissors and the adhesions, doubled after the method of Brewer, the mesentery of the appendix tied, the appendix amputated and the opening closed by means of an underhand stitch. On account of the motility of the cæcum, it was deemed advisable to follow Wilms's method of immobilizing. After that the peritoneum was closed by means of plain catgut, and the fascia by means of interrupted stitches of No. 2 chromic.

CASE II.—M. F., age 40, married.

Family history: Negative.

Habits: Smokes very little, one cup of coffee daily, a glass of whiskey occasionally.

History: Five years ago he had a severe diarrhœa, accompanied by cramps, nausea and severe pain over the region of the appendix. He cannot remember whether he had fever or not, but he had a chilly sensation, though no decided chills. After the attack subsided he went to the country, remained there for two weeks, and at the end of that time he felt very much better. However, on his return to town he had an acute attack of dyspepsia, a pain in the epigastrium radiating to the ribs on both sides, belching of gas, water brash, and bitter eructations after eating. In 1908 the indigestion became aggravated, so much so that he was afraid to eat very much. He suffered severe pain several hours after eating, was constantly belching gas, had sour and bitter eructations, a

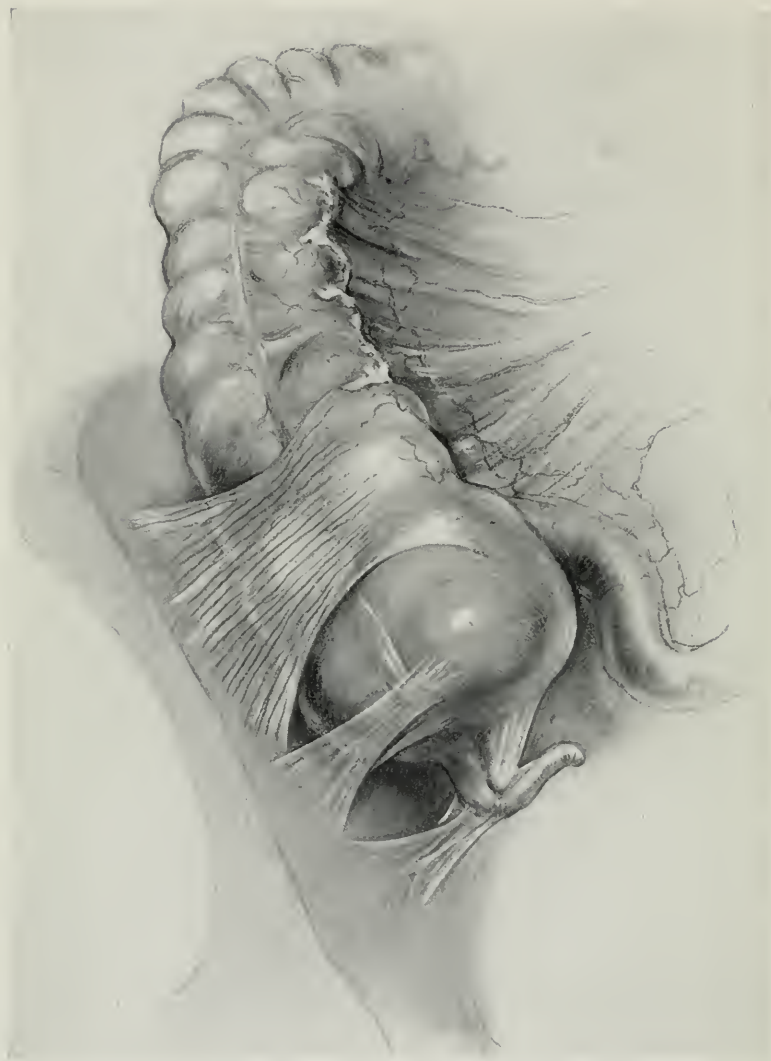
FIG. I.



Adhesions between the ileum and the appendix.



FIG. 2.



Adhesions between the caput coli and the appendix.

feeling of emptiness, yet very little satisfied him. He felt very much better for a while after eating, as this seemed to ease the pain in the small of the back, which felt as if something were broken. He would wake up at about two or three o'clock every morning and find it very difficult to go to sleep after that.

In November, 1910, he had an attack of rheumatism, for the relief of which he took a dose of salts every day. As a result he suffered from a burning around the anus, pain during and following a stool, and a feeling of unfinished business. Later he passed quantities of mucus, mixed with blood, and sometimes blood alone. The blood would continue to drop for some time after the bowel movement. His bowels were regular up to five years ago, but since then he has suffered from constipation. For one year he had great difficulty in keeping the bowels open, even with cathartics, and his stools are never satisfactory. For the past year he has suffered occasionally from severe headaches, a tired feeling, inability to concentrate, and a weakness of the muscles.

Examination: Heart and lungs normal. Ensiform cartilage, bifid, and movable. Stomach in normal position; some splashing. Appendix palpable, cæcum extended and filled with fluid. On palpation of the appendix the patient experiences pain radiating to the umbilicus, and from there into the epigastrium; also a feeling of nausea. The cæcum is thickened and rolls up under the fingers.

Proctoscopic examination: Internal ulcerated hemorrhoids. Atrophy of the mucous membrane. Prolapse of the sigmoid.

He was advised to enter the hospital, which he did. The routine preparation consisted of a warm bath the night before operation, washing off the abdomen with an alcoholic application with a sterile towel, painting with tincture of iodine.

A Battle incision was made. On opening the peritoneum the cæcum appeared in the wound; it was very much congested, movable, delivered outside of the abdomen. A portion of the ascending colon, cæcum (mobile) and appendix were covered by a cobweb adhesion. (Fig. 2.) The appendix looked fairly normal, except for an adhesion which bound it down. The cobweb adhesions were removed, and closed by an overhand suture through and through. Wound closed in the usual manner.

Uninterrupted recovery, except for a slight superficial infection. Since the operation, all symptoms have disappeared. Patient has an excellent appetite, no pain or belching after eating, and, except for a moderate degree of constipation, probably due to an obtundent sensation, is perfectly well.

This case is very instructive, and to one familiar with abdominal conditions it is quite evident what took place. Five years ago he had severe diarrhœa, accompanied with cramps, nausea and severe pain over the appendix. This was evidently an attack of acute appendicitis. This attack subsided in two weeks, and from this he dates his stomach trouble. As time went on the trouble became more aggravated, and rheumatism, three years later, was perhaps caused by this chronic infection. In consequence of taking salts constantly for the relief of the rheumatism, an intussusception of the sigmoid developed, and as a result of the intussusception, piles.

The author has noted that prolapse of the sigmoid frequently follows the continuous use of salines. Great difficulty was encountered here in making differential diagnosis, as a classical history of ulcer of the stomach was given. The diagnosis was made on the following basis:

An acute attack of diarrhœa, with pain over the appendix, followed by increasing stomach symptoms; on the fact that the cæcum was dilated and thickened, that when the appendix was palpated the pain radiated to the umbilicus and then into the pit of the stomach, and produced the counterpart of the pain experienced after eating; lastly, on the absence of occult blood from the stools and stomach contents.

The next case illustrates the fact that even an inverted Meckel's diverticulum may cause symptoms closely simulating both gallstones and gastric ulcer.

X., married, age 35, architect.

Habits: Alcohol, tea and coffee in moderation. He ate irregularly and bolted his food.

Being a man who evidently worked very hard in his profession, and who paid very little attention to his meals, he attributes the onset of his present attack to an excessively irregular mode of living.

In November, 1910, he was seen by Dr. Ives Edgerton, after having been treated for about a month for indigestion. A test breakfast showed a very high acidity, with an absence of occult blood; and washing of the stomach seven hours after regular dinner, indicated that motility of the stomach was not impaired. Dr. Edgerton then put him on the regular treatment for hyperacidity of the stomach. He improved for a while, but at the end of a month had a recurrence of the symptoms. He would have severe colicky pains, coming on at irregular intervals after eating, with belching of gas and sour eructations. The pains were acute, unaccompanied by tenderness; they were always referred to the region of the epigastrium, and as soon as the spasm passed off the patient would feel exceedingly comfortable, and have no tenderness anywhere.

During Dr. Edgerton's absence from the city, he was seen by Dr. Foot, who made a diagnosis of mucous colitis, and prescribed the rest cure. Later he was seen by Dr. Hays, and was treated for possible ulceration of the stomach or duodenum, with no relief. He was next referred to Dr. Cole, who, after a very exhaustive examination, concluded that he had adhesions between the stomach and the transverse colon, with possible diverticuli of the large bowel. An operation was advised, and agreed to. August, 1911, an exploratory laparotomy was done. The stomach, gall-bladder, pancreas and appendix were found to be normal, and the abdomen was closed. The patient made an uninterrupted recovery. Three weeks after the operation, he had a very acute attack of pain, for the first time accompanied by a rise of temperature. During the height of the attack, a tumor, about the size of a small egg, could be felt in the right iliac

region. It would become tense during the acme of the colic, and disappear immediately afterwards, leaving no tenderness. The pain at all times was referred to the epigastric region. Following one of these attacks, the patient was operated on, and a Meckel's diverticulum was discovered inverted into the bowel, causing intussusception of the bowel at that point, and a gangrene diverticulum. A resection was done with an end-to-end anastomosis. The patient had an uninterrupted recovery, and had no recurrence of the symptoms.

I am very much indebted to Dr. Edgerton for allowing me to publish this case.

As we have shown, many difficulties are encountered before a differential diagnosis can be made between duodenal ulcer and disease of the cæcum, gall-bladder and lower ileum, but with the definite modern methods at our command, by a careful history, and an equally careful subsequent study of the case, we can, in the majority of instances, come to a definite conclusion. The greater number of cases of duodenal ulcer give a history such as Mr. Moynihan has suggested, but one should always be on his guard lest he fall into error, or be too hasty in his decision. None of us is infallible, and errors of judgment are but human. The man who makes an incorrect diagnosis, after exhausting every means at his command, should not be censured. Such errors are excusable, and the physician making them is likely to benefit by his mistakes. The man who is dogmatic, positive and jumps at conclusions, without due deliberation, is the man who deserves censure.

## TUBERCULOSIS OF THE GENITO-URINARY ORGANS \*

BY ARTHUR DEAN BEVAN, M.D.

CHICAGO.

IN a discussion on tuberculosis of the genito-urinary organs I want to remind you that our knowledge is of comparatively recent date. It is hardly necessary to refer to the fact that it was only in 1882 that Koch demonstrated to us the *Bacillus tuberculosis*; and until, I should say, about 1885 we regarded tuberculosis of the urinary tract as inoperable, as difficult of diagnosis, and as a field that was more interesting to the pathologist than to the clinician. I should date our present knowledge from about 1890, when it was very definitely demonstrated that we have in genito-urinary tuberculosis two distinct types, the genital and the urinary cases, which up to that time had been grouped together as one inseparable pathological condition. I can remember very well seeing cases before that time that were regarded as being tuberculosis primarily of the epididymis, and then extending to the bladder, and from the bladder to the kidney. We know now that the condition is very different, and our conception of genito-urinary tuberculosis is very much the same as our conception of tuberculosis of the hip-joint. In a child five or six years of age, with a tuberculous lesion of the hip-joint, we regard the disease as being an infection through the circulation. We believe that in the majority of cases there is first an infection through the air-passages or alimentary tract; that later lymphatic glands are involved, and that, in addition to the involvement of the lymphatic glands, we have the escape of tubercle bacilli into the general circulation. Some of the tubercle bacilli are carried to the hip-joint, to a point close to the line of epiphyseal cartilage, and there meet conditions favorable for their development. We are not astonished to find but one hip involved in this hæmatogenous infection. We take it for granted that but few bacilli have found their way in the circulation, and that these have unfortunately found conditions in the one hip-joint favorable for their

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\* Read before the South Side Branch of the Chicago Medical Society, November, 1911.



development. The same is doubtless true of tuberculosis of the genito-urinary tract.

There are in the genito-urinary tract three positions which seem to favor the development of the tuberculous process when the tubercle bacilli are brought to these organs through the circulation. These are: (1) the kidney, (2) the epididymis, and, (3) the prostate. On analysis of any case you will find practically without exception that one of these three anatomical locations is the original primary focus of the condition. I suppose that one can say, without much hesitation, that these foci in the kidney or epididymis or prostate can be called primary in this sense—that they are the overshadowing locations of tuberculosis in the particular individual. The individual may not present clinically any other gross evidence of tuberculosis, but we should keep in mind the fact that they are not really primary, but that they are secondary to some invasion through the respiratory tract or the alimentary tract, although one must admit the possibility of such a condition as this either in hip-joint tuberculosis or in tuberculosis of the kidney; *i.e.*, that the tubercle bacilli may find their way through the respiratory tract or the alimentary tract directly into the circulation, within a limited time, without producing first any involvement in a lymphatic gland. With this conception, then, of tuberculosis of the genito-urinary tract, let us discuss, first, tuberculosis of the kidney.

Tuberculosis of the kidney, like tuberculosis of the hip, is usually a primary affair. I should say that you would probably find both hips involved in tuberculosis in possibly one case out of twenty, thirty, or fifty—I am not sure of the accepted statistics. I am rather inclined to think that in about the same proportion of cases both kidneys are primarily involved in the tuberculous process. The involvement in the kidneys is usually a fairly extensive one.

We have in our collection a large number of tuberculous kidneys, and we seldom find a kidney in which the process is limited. I think Fig. 1 will represent a good type of the ordinary case. When you look at it you will find that the process, although it is much more marked at the lower pole of the kidney, is very distinct, both in the capsule behind and on the split surface, throughout the entire kidney. That should be borne in mind, because we suffered in some of our early work from the conception that we have in tuberculosis of the

kidney frequently to deal with a limited process, and many of us resort to resection of the kidney with the hope of curing the disease. One can take this lesson from our pathological specimen, that, clinically, when we operate upon these cases they are so frequently riddled with tuberculosis that we can eliminate from consideration resection of the kidney as a means of cure. The tuberculous process very rapidly extends along the ureter.

Fig. 2 is a very beautiful specimen, showing the wide extent of the tuberculous process along the ureter. I think one can accept, as a rule, the fact that tuberculous process extends along the ureter and the ureteral orifice in the bladder and soon involves to a greater or less extent the bladder mucosa.

What is the natural history of tuberculosis of the kidney? I think one can say that in a limited number of cases a cure is effected. How is that cure brought about? Usually by the obstruction of the ureter and by the kidney becoming a sac filled with tuberculous débris. I have in several cases operated and found a thick fibrous sac filled with tuberculous débris without a single vestige of kidney material, the ureter being quite distended, extending evidently down to the bladder. As no tubercle bacilli appeared in the urine there was probably a complete obliteration of the ureter. The patient experienced no symptoms whatever from that kidney. Undoubtedly these cures resemble very much the cures from psoas abscess in Pott's disease of the spine; there is a walling off of the tuberculous débris and eventually absorption of the abscess or its encapsulation in such a way as to prevent the condition giving rise to symptoms.

I have personally never seen a case of kidney tuberculosis which has healed by the development of cicatricial masses in the kidney itself. That they do occur I believe has been shown, but they are rare.

Where the individual does not go on to this cure by obliteration of the kidney the tuberculous process usually involves extensively the bladder and later other portions of the body. I suppose we must accept the possibility of an infection by way of the bladder from one kidney to the other. I am inclined to believe, however, it is not common, and that infection of the second kidney is hæmatogenous. We have infection of both hips in hip-joint disease, but we cannot imagine any infection from one hip to the other.

FIG. 1.



Tuberculous kidney, showing wide-spread involvement.

FIG. 2.



Tuberculous kidney, showing the process extending along the ureter in the form of miliary deposits.



The diagnosis of kidney tuberculosis to-day is not nearly as difficult as it was a few years ago. The first symptom is frequently of urination. The bladder symptoms stand out prominently: pain above Poupart's ligament, occasional hemorrhages, occasional pain in the region of the kidney. Pain in the kidney involved is by no means a constant symptom. As a rule, however, we do have pain and discomfort in the kidney, and, occasionally, severe renal colic, meaning usually obstruction of the ureter with blood and tuberculous matter, and a marked increase in the intrarenal tension. In a limited number of cases we find a tumor. The diagnosis is usually made by a process of exclusion, this being done by means of the X-ray, the determination by cystoscopic examination of the condition of the opening of the orifice of the ureter in the bladder, and, best of all, by discovering, by means of the ureteral catheter, the presence of abnormal urine from one side or occasionally from both sides, and the demonstration of tubercle bacilli in the urine.

Within the last two years we have been finding tubercle bacilli fairly constantly in our cases of kidney tuberculosis. We used to regard that as an exceedingly difficult thing, and I remember very well making the statement a few years ago that we did not find tubercle bacilli in more than fifty per cent. of our cases, but I do not hesitate to say now that we find tubercle bacilli in ninety per cent. or more of cases of definite kidney tuberculosis. Occasionally one will have to depend upon animal inoculation to demonstrate the tuberculous character of the lesion.

As to the treatment of kidney tuberculosis, I think we can be fairly confident that in primary tuberculosis of the kidney, if the case is seen early and when it is limited to one kidney and we can make that diagnosis with certainty by means of catheterization of the ureter, the life of the patient is very much safer with an early nephrectomy than by any other plan of treatment.

In the last year or two there has been a reaction against nephrectomy for kidney tuberculosis, which, I believe, is entirely unwarranted. A nephrectomy for early tuberculosis gives a low mortality, and the patient has a very much better prospect of permanent cure than is afforded by any other means we have at our command.

What shall we do with the ureter in our nephrectomies? I am not at all satisfied with the way we handle the ureters. I dislike very



much to make a very extensive and sweeping operation of exposing the entire ureter down to the bladder, and I have been recently handling them after a nephrectomy by injection with carbolic acid and by ligating their ends. I have tried bringing the ureter out and injecting it with iodine and with iodoform emulsion and with alcohol—and we have had some successful cases with these methods—and for a time I was inclined to believe that the use of alcohol, bringing the ureter out and stitching it in the wound was the preferable method. I am still undecided as to the best procedure. The weak point that still makes unsatisfactory our nephrectomies for tuberculosis is the handling of the ureters.

In regard to other methods of treatment for kidney tuberculosis, the fresh-air and outdoor treatment, along with the proper employment of large quantities of milk and eggs, which a patient with lung tuberculosis receives, are of enormous value in all cases of surgical tuberculosis.

I am a little doubtful about tuberculin. I have used it, but I am not at all enthusiastic about it. I think it is difficult to demonstrate the relationship between the use of tuberculin, either by the Wright method or by greater doses,—and the resulting cure. I think the evidence furnished by orthopædic surgeons must be considered in this matter. In handling large numbers of cases of tuberculosis they, as a group, are not very favorably impressed, I take it, by the value of tuberculin. I think tuberculin should be used, however, in cases of kidney tuberculosis. When both kidneys are affected undoubtedly tuberculin and the fresh-air treatment would seem to be the most intelligent way of handling the case.

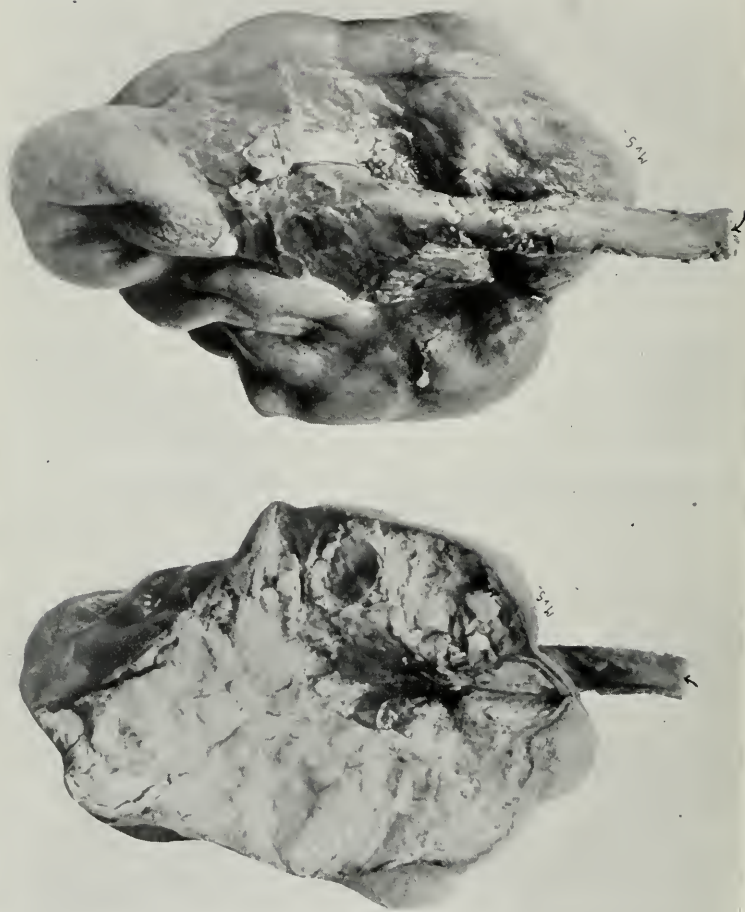
As to the final outcome, Dr. Louis Schmidt made a rather interesting statement to us at a recent meeting of the American Urological Society when discussing this matter. He stated that he was impressed with the fact that few of these cases of nephrectomy for bladder tuberculosis were cured in the sense that all of the tubercle bacilli disappeared from the urine. He quoted some evidence that had been recently offered to show that even where there was an apparent cure, and the patient was well and strong, had regained his weight, and had no symptoms, careful examination of the urine often showed tubercle bacilli. I was very much impressed with that statement, and since hearing it in two cases in my own work I have been able to find

FIG. 3.



Ordinary forms assumed in tuberculosis of kidney. In the lower figure the process is more marked to the right, but there are distinct lesions in the capsule and cut surface throughout the entire kidney

FIG. 4.



Tuberculous kidney, with occlusion of ureter.

tubercle bacilli in the urine, even though the patient in one instance had no symptoms. In the other case the nephrectomy wound had entirely healed, and the child was strong and well apparently in appearance, but she had an incontinence which gave her a good deal of trouble, though it did not seemingly interfere with her general health.

I asked Dr. Kretchmer to make a cystoscopic examination, which he did, and found that the bladder was contracted down so that it only held about an ounce and a half of urine. She had extensive bladder tuberculosis extending from the ureter of the infected kidney, in spite of which she was apparently well, so far as her general condition was concerned.

The doctrine, as you know, has been, and we have preached it emphatically, that the bladder mucosa cleans up quite constantly after the removal of the primary focus. I believe we must look a little more carefully into this matter, and I shall not be at all surprised to find that in quite a large proportion of these cases the bladder mucosa has not cleaned up entirely, even though the case is apparently clinically cured.

In regard to tuberculosis of the bladder from the kidney, I will simply state that it is a descending infection. We all accept the position now that primary tuberculosis of the bladder is exceedingly rare, if it ever occurs, and it is either due to a descending infection from the kidney or an ascending infection from the epididymis, vas deferens, and seminal vesicles, or the prostate. I have never seen anything do any good in cases of bladder tuberculosis except rest, and that is a difficult thing to give a bladder in cases of tuberculosis. If you drain such a bladder suprapubically you often get a tuberculous fistula, with extension of the process along the fistulous tract. The most satisfactory cases of tuberculosis of the bladder I have seen have been drained into the vagina, and in a few of these instances a good deal of relief has been given to the patients. From my own experience of bladder tuberculosis, I think the cases are good ones to leave alone, so far as any direct interference is concerned. I am inclined to believe that these are cases where we should make the patients as comfortable as possible and trust largely to the fresh-air treatment and to the use of tuberculin.

In connection with the use of tuberculin, we have had several



cases of bladder tuberculosis where, in addition to the treatment, the autogenous vaccines from the colon have been used with apparently a great deal of benefit. That point should be emphasized in handling cases of tuberculosis of the bladder.

Tuberculosis of the epididymis is exceedingly common, and resembles in its pathology the like infection of the oviducts. It would be much more frequently found, if searched for as a routine measure in diseases of the external genitals. In these cases the infection is hæmatogenous.

Tuberculosis of the epididymis should be handled early by surgical intervention. In tuberculosis of the epididymis and of the vas deferens these structures lose their function. I do not believe any cure of a tuberculosis of the epididymis or of the vas results in the restoration of function. By a comparatively simple and very safe procedure the epididymis and vas can be removed without depriving the patient of the benefit of his testicles, so far as internal secretions are concerned, and I believe we should take the position, in tuberculosis of the epididymis and vas, that the best procedure is early radical removal of the infected tissues. I should say forty per cent. or more of these cases can be cured by early surgical treatment. That has been our experience. I would add, however, that without any question these patients with tuberculosis of the epididymis and of the vas should be given the same benefit of outdoor and fresh-air treatment as cases of tuberculosis of the lung. I have seen very startling cures from change of climate, and the benefit of fresh air and feeding, where patients have refused operative interference. We should add to this the use of tuberculin in minute doses.

When the process has extended to the seminal vesicles we have a very serious condition. I have operated on a few of these deeply-seated cases of genital tuberculosis, and I have not improved any of the patients. I doubt whether it is good surgery, in the light of our present knowledge, to operate on cases of tuberculous involvement of the seminal vesicles. Of course, when we have a mixed infection with abscess, drainage should be resorted to. I am inclined to handle cases without mixed infection as we do cases of tuberculosis of the lung.

Tuberculosis of the prostate is sometimes primary. It is a rare thing. I have operated on three cases for tuberculosis of the prostate

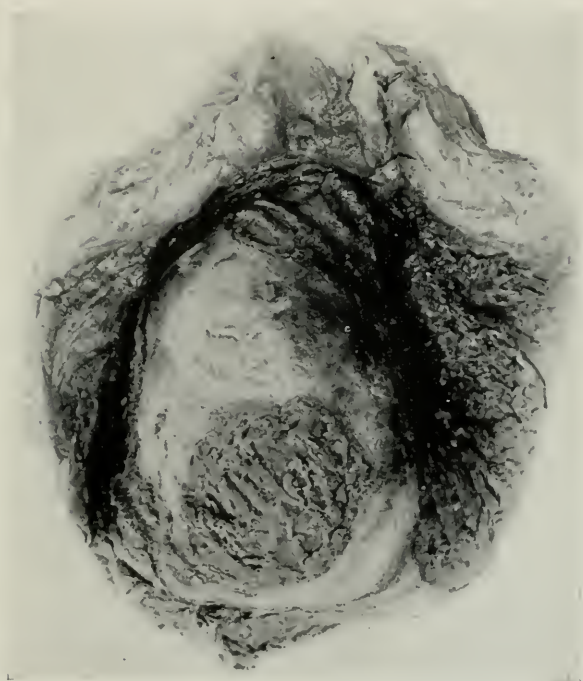


FIG. 5.



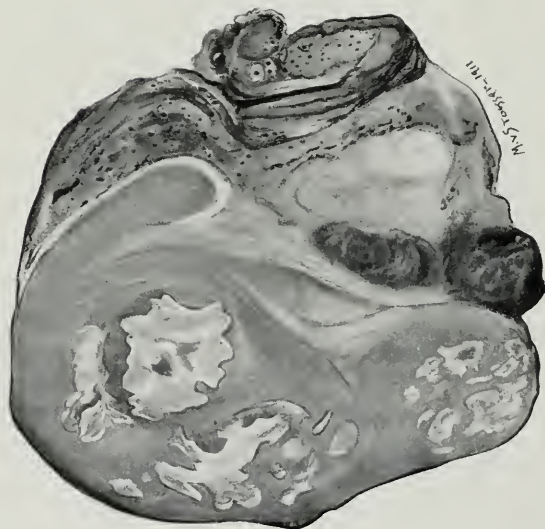
Tuberculosis of prostate.

FIG. 6.



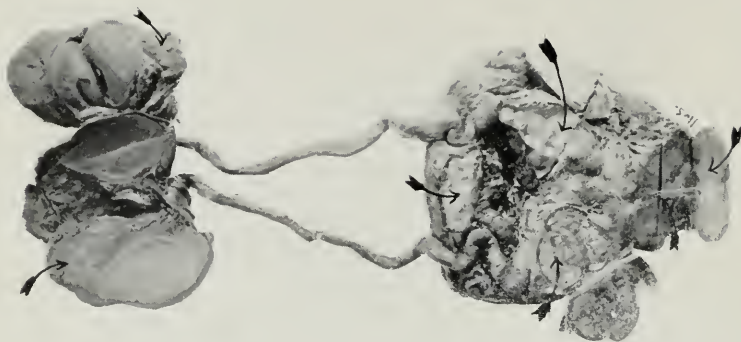
Tuberculosis of the urinary bladder, with thickening of the bladder-wall and great contraction.

FIG. 7.



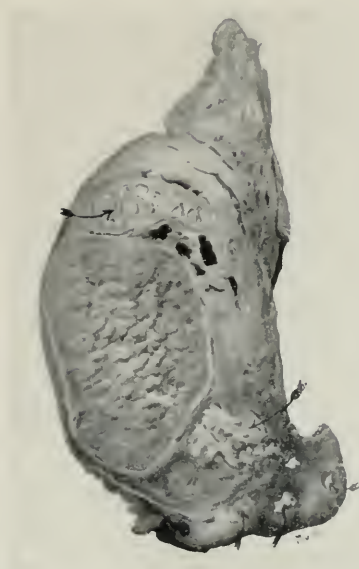
Tuberculous of the testicle.

FIG. 8.



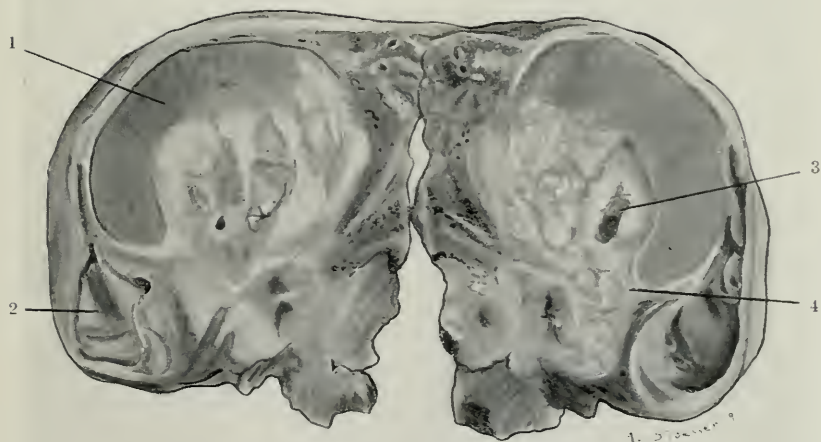
Tuberculous of epididymis, extending to the vasa deferentia, seminal vesicles, and prostate.

FIG. 9.



Chronic caseous tuberculosis of the epididymis.

FIG. 10.



Tuberculosis of the testicle and epididymis. 1. Normal testicular tissue. 2. Cavity filled with clear fluid (hydrocele). 3. Tuberculous area. 4. Line between testicle and epididymis.



by prostatectomy. I think one can safely say that it is very much more rare than tuberculosis of the epididymis or of the kidney. My own conception of tuberculosis of the prostate would be, that if we could find a case where the lesion is distinctly limited to the prostate and we could make an early complete removal it would be the better procedure. Unfortunately, however, in most of these cases the process has widely extended before we operate upon the patient, and on that account we should regard cases of tuberculosis of the prostate, so far as treatment is concerned, as belonging to the same group as the cases of lung tuberculosis; that they should be handled by tuberculin and by the general hygienic treatment for tuberculosis.

I have brought from our clinical museum a number of specimens from cases of tuberculosis of the genito-urinary tract. I will show you kidneys which are involved in the tuberculous process. I want to repeat the statement which I made, that these illustrate quite clearly, without exception, the widespread involvement (Fig. 3).

In examining these specimens it is seen how impossible it would be to consider anything except a nephrectomy.

In addition to the kidney specimens we have some interesting specimens of kidney tuberculosis with occlusion of the ureter (Fig. 4).

Here is a specimen of tuberculosis of the bladder, ureter, and kidney in a case of tuberculous meningitis. This specimen was removed postmortem.

In the next specimen almost the entire prostate is involved in the tuberculous process (Fig. 5).

Here is a specimen of chronic tuberculosis of the urinary bladder, showing great thickening of the bladder wall, and great contraction (Fig. 6).

Here is a specimen of tuberculosis not only of the epididymis, but also of the testicle. Here is another specimen of chronic tuberculosis of both the testicle and epididymis. Clinically, if cases of tuberculosis of the testicle are seen early, the tuberculous process is limited largely to the epididymis (Fig. 7).

Here is a case of ascending genital tuberculosis, beginning in the epididymis and extending to the vas deferens, seminal vesicles, and prostate (Fig. 8).

Here are two more specimens illustrating tuberculosis of the epididymis (Figs. 9 and 10).



In conclusion, I should like to make the following statement: We are in a position to-day of looking at this entire group of cases as a borderland where the internist and general surgeon and genito-urinary surgeon can very well meet on common ground. It is not an intelligent thing for the internist to say that these cases of genito-urinary tuberculosis should be handled with tuberculin and with fresh-air and outdoor treatment and not in an operative way. On the other hand, it is not proper for the surgeon to take the position that they should all be handled by operative measures. Enough light has been thrown upon this subject to show the great value of both surgical and medical treatment, and undoubtedly some of these cases can be handled by medical management alone, some largely by surgical management alone, but most of them must be regarded as cases which can be handled properly by only combined medical and surgical treatment.

# CANCER OF THE HOLLOW VISCERA OF THE ABDOMEN, WITH SPECIAL REFERENCE TO DIAGNOSIS \*

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ACCORDING to the mortality statistics of the census of the United States for 1910, cancer ranks sixth on the list.<sup>1</sup> It is estimated that there are now in the United States 225,000 cases, this being an increase of 8,000 over 1908. There were 75,000 deaths from cancer in the United States in 1910. This means that annually one person out of every 1,200 in the United States dies of cancer.

According to Rodman,<sup>2</sup> one-third of all cancers originate in the stomach. Stierlin<sup>3</sup> says of all deaths from cancer three and one-third per cent. are due to cancer of the rectum. Without going into further detail, we are probably warranted in saying that between forty and fifty per cent. of all cancers originate in the hollow viscera of the abdomen. In this estimate cancer of the urinary bladder is not included.

A vast majority of patients with cancer of the hollow viscera present symptoms of either obstruction or ulceration or both, when they first consult the surgeon. This means that they come to the surgeon late—too late to achieve through surgery all the chances which it can offer of permanent cure. The deaths from cancer of the hollow viscera are very largely due to late diagnosis.

When anæmia, cachexia, and tumor are present, the disease is in an advanced stage. E. C. Hart<sup>4</sup> regards a normal antitryptic blood-content as of great value in excluding a diagnosis of cancer. A raised antitryptic content, however, is present in measles, scarlatina, syphilis, and several other diseases, but the test seems to be of value in distinguishing between an innocent and a malignant neo-

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\* Read at the annual meeting of the Western Surgical Association, Kansas City, December 18, 1911.

plasm. Authorities differ as to the value of the hæmolytic test. The glycyl-tryptophan test involves too many sources of error to be of practical value. The value of the nitrogen content of the urine is still undetermined, according to E. Salkowski.<sup>5</sup> Where the diagnosis question is between gastric cancer and ulcer the finding of Gram-positive stools indicates that the case is one of cancer, according to P. H. Brown.<sup>6</sup> John B. Deaver<sup>7</sup> says there has not yet been discovered any specific serum or hæmolytic reaction in patients suffering from cancer.

The gastroscope has as yet given us no practical aid in the diagnosis of gastric cancer. I have but little faith in the practical value of the X-ray and bismuth test as an aid in the diagnosis of cancer of the hollow viscera. This test will demonstrate strictures and ulcers, but will not tell anything concerning their malignancy or otherwise. In other words, this method of examination will only demonstrate old surgical lesions. The routine use of the sigmoidoscope by the general practitioner as well as by surgeons will result in a material improvement in the statistics of cancer of the rectum and sigmoid. Digital examination of the rectum with the patient standing will enable one to reach growths with the finger that can not be detected with the patient in the recumbent position.

In the past too much attention has been paid to the so-called cancer age. The duration of cancer has been, and still is, in my opinion, much underestimated. Kocher says that most of the cases of rectal cancer are of two or more years' duration when he sees them first. This is my experience in all cancers of the hollow viscera, excepting the appendix vermiformis. In the Rostock clinic four cases of rectal cancer out of a total of 115 occurred in patients between fourteen and seventeen years of age. In my own work I have seen many cancers in patients under thirty years of age. None of these, however, have been cancers of the hollow viscera. Schnitzler's<sup>8</sup> observations are interesting, but lack confirmation, and, even if confirmed, offer little hope of help in early diagnosis. He says that cancer of the stomach should always be suspected when the lymph-glands above the clavicle and in the axilla are large, hard, and not tender. Still more suspicious of cancer of the stomach, he says, is an infiltration in the pouch of Douglas. He has not seen meta-

stasis in the pouch of Douglas in connection with cancer of the other hollow viscera, but has seen it in one case of pancreatic cancer.

Why is it that the prognosis in cancer of the appendix is relatively good? In my judgment, it is because the rule is to open the belly on the slightest provocation from symptoms in the right lower quadrant of the abdomen, and to remove the appendix on suspicion. Voeckler<sup>9</sup> accounts for the relatively good prognosis in cancer of the appendix on the ground that inflammation of the appendix makes its presence known very early. This is only a partial statement, however. The fact is that we not only recognize inflammation of the appendix early but that we remove it as soon as we know that it is inflamed.

Mistakes in diagnosis are frequently made even after an exploratory operation. In a paper read at the recent meeting of the Southern Surgical and Gynecological Association on the subject of "Cancer of the Rectum and Lower Sigmoid" I referred to four cases in which innocent disease of the colon was mistaken for malignant disease. One each of these cases occurred in the practice of Drs. McCosh, Brewer, Stimson, and the writer. The difficulty of diagnosing between malignant and non-malignant conditions of the bile-tracts even with the abdomen laid open is well known. Rolleston<sup>10</sup> reports two such cases in his own practice. In one malignancy was supposed to exist and did not, and in one the trouble was thought to be inflammatory and proved later to be malignant. Twice I have mistaken benign changes consequent upon cholelithiasis for malignant disease. In one case the abdomen was closed after simple exploration. A postmortem examination made some months later developed the fact that the trouble was inflammatory and consequent upon a large gall-stone lodged in the ampulla of Vater. This case occurred twelve years ago. To-day I would in such a case drain the liver through the abdomen and later attempt a radical cure. The second case is as follows:

CASE I.—Mrs. F., aet. 62, housewife, American, was admitted to Hope Hospital, August 3, 1910. Family history: One sister died of cancer, one of tuberculosis and one of "uterine tumor." Previous history: Negative save that she frequently suffered from "indigestion." Present trouble began about three months ago with severe pain in right side and back, in liver region, accompanied

by nausea and constipation. She has had several similar attacks since the first one, the last occurring two days ago. She has been in bed two weeks.

Examination showed slight jaundice, considerable loss of flesh, temperature normal, pulse 90. There was a tender mass in the gall-bladder region. Operation revealed a gall-bladder distended with mucoid fluid slightly bile-stained and containing a large mulberry stone, which was removed. The liver was distinctly nodular and hard. The gall-bladder was drained. The family was told that the trouble was probably malignant and that only temporary relief was to be expected. Now, fifteen months after the operation, the patient is well.

An instructive case wherein non-malignant disease of the stomach was at the time of operation considered to be malignant is the following:

CASE II.—Mr. B., aet. 44, merchant, single, was admitted to Hope Hospital, April 19, 1907. Family history negative. Previous history: Had suffered ten years with attacks of indigestion. Three attacks were characterized chiefly by recurring pain and discomfort in the epigastric region. Vomited at times, but never vomited blood. Has had streaks of blood in the stools a few times. Recently the discomfort has been continuous and the vomiting more frequent. He has been confined to the house ten days and to bed four days.

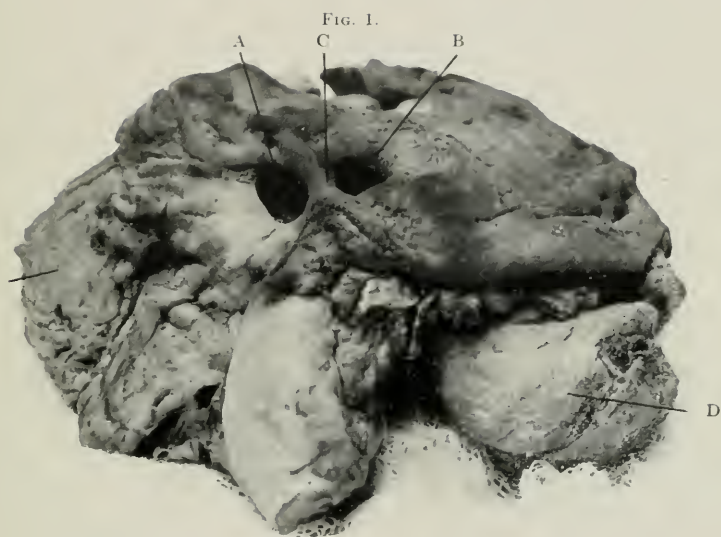
Examination: Patient is much emaciated and cachectic. There is a mass, somewhat tender and very hard, in the region of the pylorus. Operation, April 20, 1907. The usual incision revealed an irregular hard mass in the pyloric end of the stomach which was adherent to everything in the vicinity. The lymphatics were enlarged. A posterior gastrojejunostomy was done and the opinion given that the trouble was malignant and that the operation would only be palliative. At the present writing the man is in perfect health, now four years and eight months after the operation.

CASE III.—Mr. R. H. C., a married farmer, 47 years old, was admitted to the hospital October 25, 1911. Family history: Two paternal aunts and one son died of tuberculosis. Previous history: Typhoid fever twelve years ago. Has been subject to frequent bilious attacks all his life. Lately the intervals between these attacks have grown shorter. Fifteen months ago during one of these attacks two gall-stones were passed. Vomitus is green, slimy, and bitter. Never vomited any blood. Has lost fifteen or twenty pounds in the last few months. Present history: This attack came on with severe pain in epigastrium and vomiting "slime and bile." Bowels constipated.

Examination: Small, poorly nourished, medium complexion. Lungs and heart negative. Abdomen is scaphoid and the walls very thin. An indistinct mass was felt above and slightly to the right of the navel. A probable diagnosis of cholecystitis was made.

Operation, October 26, 1911. Vertical incision through right rectus. Pylorus and duodenum were found adherent to under surface of the liver and to the gall-bladder. Pylorus was patulous and stomach walls apparently healthy. The head of the pancreas was enlarged and adherent to the posterior aspect of the pylorus. The gall-bladder was opened and found to contain thick, stringy,





Photograph of specimen removed from Case 3, showing pylorus opened on anterior aspect. At *A* and *B* are seen the two openings of the ulcer, which openings communicate beneath the pyloric band *C*. *D*, portion of pancreas.



dark bile containing many granules, but no stones were found. Under the impression that I was dealing with an infection of the bile-tracts which had caused a pancreatitis, the gall-bladder was drained and the wound dressed in the usual way. The patient did well for five days, when he complained of severe pain in the stomach and vomited. The vomitus contained some changed blood, and on the same day the patient had two rather copious dark-brown liquid stools. The pulse now became weak and rapid. Within twelve hours of the onset of this attack the patient was feeling quite comfortable and the pulse had dropped from 136 to 100 and had improved in character. From this time on the patient vomited occasionally. Sometimes only the food taken would be vomited and sometimes there would be blood in the vomitus. A day or two or a longer time would elapse during which the patient was quite comfortable and took food without distress. On the eighth day after the operation it was noted that there was some blood on the dressing. Total rest of the stomach seemed to be of no avail. The patient died on the twenty-fifth day after the operation from a severe hemorrhage.

Postmortem examination revealed what might be termed a saddle ulcer of the stomach and duodenum, with one skirt of the saddle in the stomach and the other in the duodenum, and the two skirts communicating beneath the pylorus. This pyloric band which crossed the ulcer was perfectly soft, flexible, and apparently normal. The stomach and duodenal walls up to the margin of the ulcer seemed perfectly normal. The pyloric end of the stomach and the commencement of the duodenum, together with the head of the pancreas, were removed *en masse*, and before the pylorus was opened I attempted to pass my finger through it, when I discovered apparently two pyloric openings, one, in front, normal, and one, posterior, strictured and non-distensible. Not until the specimen was split along the anterior wall was this finding made clear. The cavity of the ulcer was filled with a blood-clot, which on removal was dumb-bell in shape. The floor and side walls of the ulcer were formed by the pancreas. The head of the pancreas was enlarged and hard, and the pathologist's report was "interstitial pancreatitis with some autodigestion." Figure 1 is made from a photograph of the specimen and will give a clearer understanding of the conditions.

*Remarks.*—Looking backward, one sees perfectly clearly that this patient's belly should have been reopened and a posterior gastro-jejunosomy done as soon as he rallied from its first hemorrhage. The advisability of a second operation was discussed with my *confrères*, but it was not done because at the first operation the pylorus was found patulous and distensible and the head of the pancreas enlarged and hard. Moreover, there were also demonstrated at that time a cholecystitis and pericholecystitis. These findings, taken together with the previous history and the first five days of the post-operative history, lead me to conclude that we could rule out gastric ulcer and probably had to deal with a pancreatic trouble

malignant in character. This opinion seemed strengthened also when on the eighth day after the operation the dressings were found quite bloody. I want to say here that this is to be taken as an explanation pure and simple of how I was led into grievous error, and not as an excuse or plea for leniency. In fact, one of the chief objects of this paper is to do what I can to make it unnecessary for others to learn by experience, for "'tis true, 'tis pity, and pity 'tis 'tis true," but the fact remains that when one says he has learned by experience it is only another way of saying that he has learned from his mistakes.

#### CONCLUSIONS

1. The symptoms commonly regarded as sufficient to warrant a diagnosis of cancer of the hollow viscera are such as are not manifest until the favorable time for surgical interference has passed.

2. Success in the treatment of cancer of the hollow viscera depends largely upon its early recognition.

3. It is often unsafe and unwise to make a diagnosis between malignancy and benignancy without the aid of the microscope.

4. The way to improve our results in the surgical treatment of cancer of the hollow viscera lies in the direction of earlier cœliotomy and immediate microscopic examination of the specimen.

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- <sup>5</sup> *Amer. Jour. Med. Sciences*, 1911, cxli, 443.
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- <sup>9</sup> *Jour. Amer. Med. Ass.*, 1908, li, 355 (*Arch. f. Clin. Chir.*).
- <sup>10</sup> ROLLESTON: "Diseases of the Liver," etc., 1905, 634.

## EPILEPTIC MASKS AND THE DIFFERENTIAL DIAGNOSIS OF EPILEPSY \*

BY WM. T. SHANAHAN, M.D.

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THE diagnosis of idiopathic epilepsy is not infrequently delayed for many years, owing to various reasons, among which are incompleteness of knowledge and improper interpretation of the symptoms on the part of the attending physician, mildness of type of the condition, and coexistence with other disorders which overshadow the phenomena of epilepsy.

On the other hand, the diagnosis of idiopathic epilepsy is made incorrectly where some of the following conditions are the underlying causative factors of the seizures: brain tumor or abscess, dementia præcox, general paresis, uræmia, eclampsia, etc.

Many careful observers have concluded that there is no one pathognomonic sign by means of which epilepsy can be absolutely diagnosed. Each case must be studied with great care and all of the symptoms considered before a positive diagnosis can be arrived at.

The essential feature of a true epileptic seizure is supposed to be a *sudden* loss or impairment of consciousness, this symptom recurring at varying intervals in those having genuine epilepsy. We all are aware, however, that this symptom is also seen in other conditions which are not designated as epilepsy.

In many cases, especially in hospital practice, it is exceedingly difficult to obtain anything like a satisfactory personal history of the patient. We must always bear in mind that one convulsion or a series of convulsions is not proof conclusive that the individual so afflicted has an idiopathic epilepsy. In this connection it would be well to consider the statement of Gowers: "Every fit is in part, at least, the result of those which have preceded it, and in part a cause of those which follow it." This one convulsion or series of con-

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\* A lecture delivered at Fordham University School of Medicine, New York City.



vulsions may be but the precursor of a great number of convulsions which are to occur during the lifetime of the patient.

The diagnosis of epilepsy cannot be absolute unless the physician himself sees the seizures or a proper trained nurse observes and reports for him. A hastily-given positive opinion should be carefully guarded against, although at the same time one should not dwell too lightly upon the importance of symptoms analogous to those of epilepsy.

The non-recognition of the meaning of *petit mal* seizures is a very frequent error on the part of parents and of physicians as well. The assurance is made that the "spells" are so slight that they surely amount to nothing; they come from the stomach; they will pass off when the child is older, etc. Many writers on the subject have called attention to this non-recognition in many instances of epilepsy until it has been established for many years. The child who has convulsive attacks, be they so-called "worm fits," "teething fits," or what not, may have these disappear for a period, but the convulsive tendency has been established and simply remains to be again brought to light at a later period in the life of the individual. *Too much stress cannot be laid on the importance of this one phase of the subject of epilepsy and epileptiform phenomena.*

Any epileptic may have recurring phenomena not accompanied by interference with consciousness: myoclonic symptoms, epigastric sensations, headache, etc. In a considerable number of epileptics the change in the conscious state is so transient as to escape notice unless the observer is looking directly at the patient at the time of the seizure.

The cry, the fall, the sequence of tonic and clonic convulsive movements, the wetting of the clothing or bed, the biting of the tongue, may all be observed during any complete convulsion, but such is not necessarily what we call epileptic in nature. When such a group of phenomena recur over a period of years, especially in part during sleep, we call the condition idiopathic epilepsy, which is simply a term for a group of symptoms of which the exact underlying factors are unknown. Strictly speaking, idiopathic epilepsy means a chronic, progressive disorder characterized by recurrent, abrupt attacks of impairment or loss of consciousness, with or without convulsive phenomena, and accompanied by mental and oftentimes physical

FIG. 1.



Complete convulsive seizure, tonic verging into clonic stage.

FIG. 2.



Clonic stage of complete convulsive seizure.

deterioration, and of whose definite causative factors we are more or less ignorant.

A history of having repeatedly fallen from bed during sleep; of having severe headaches on arising, for which no apparent cause can be discovered, this recurring with more or less periodicity; a feeling of inexplicable fatigue and heaviness and the sensation of having been beaten, make us form the opinion that such an individual is epileptic. In addition to this, if the inner surface of the cheeks and lips or the tongue is lacerated or sore and some blood is repeatedly found on the pillow, there can be but little question of the patient's disorder.

The differential diagnosis between epilepsy and hysteria is oftentimes a very difficult one to establish. The two disorders are based upon a hereditary degenerative foundation and have certain symptoms in common: *e.g.*, periodic paroxysms showing many analogous features. In the epileptic, both male and female, many hysteroid phenomena are commonly seen. There are, however, ordinarily essential points of difference which mark out distinctly the exact disorder present in each instance.

In the complete, severe epileptic seizure there is the definite sequence of symptoms: cry, fall, loss of consciousness, tonic convulsion, clonic convulsion, stertor, stupor, and sleep. The onset is sudden, the epileptic frequently falling and receiving serious injuries. In hysteria the onset is gradual, the patient never receiving injury, care being taken to avoid such. In the typical epileptic seizure, the loss of consciousness is complete, with absence of corneal reflexes; in hysteria, on the contrary, there is at most only an impairment of consciousness, the patients can be easily aroused by irritation of the skin, touching the cornea, by shaking or talking to them in a loud tone. In the hysterical seizure, the tonic spasm is accompanied by opisthotonus, emprosthotonus, or attitudinizing. They may scratch, bite at, or strike at those about them; such actions never occur in the true epileptic seizure. In place of the stertor and coma of the epileptic attack, the hysterical individual may immediately arise and walk about as if nothing had occurred. The elevation of temperature, the exhaustion paralysis, the exaggerated kneejerks, the ankle clonus with the Babinski phenomena, and the automatism are sequelæ of the epileptic but not of the hysterical seizure.



A true hysterical seizure may and does occur in an individual who also has genuine epileptic seizures. Atypical attacks of the two disorders resemble one another more closely than do the typical. To show some of the difficulties encountered in formulating a definite diagnosis, the following case is of interest:

CASE I.—Mary C. G. (2555); admitted to the Craig Colony August 31, 1908; age 35 years. Family history as obtained negative. Present symptoms said to have begun at the age of 7 years, assigned cause scarlet fever, a possible complication of which was a meningitis.

She complained of frequently feeling tired, having frequent headaches accompanied with nausea, pain and tenderness over the dorsal region, especially the latter since falling from a carriage some 7 years before. Attacks of dizziness also complained of. When told to walk, she said she could not do so, but after insisting she stood on her feet and fell down, being careful, however, not to injure herself in doing so. Finally she walked around the room several times with her legs planted far apart, feet everted and rotated inward. Knees bowed inward. Gait unsteady. It was said by her brother and the nurse in the cottage that she could not walk, but if left alone she apparently walked without difficulty. There was no apparent limitation of movement, and coördination, when lying in bed, was good. Patient's brother, who brought her to the Colony, said she had perverted sexual habits, was quarrelsome and erratic. Following her admission to the Colony, she was observed as having some pseudomyoclonic jerks. She would cry out, throw her extremities about and apparently go through a typical hysterical seizure. She never injured herself in any manner. Later at times she was able to be up and walk about, and again she would make the various complaints above mentioned.

After remaining at the Craig Colony for two years, her condition improved considerably, so she was able to walk about out-of-door without assistance. On August 29, 1910, she had a mild seizure at 10:45 A.M. Following this she remained lying down for some minutes. One-half hour later a nurse discovered she was dead. Tongue had not been bitten, no urine or feces voided, face slightly cyanotic, lips very blue, pupils equal and dilated moderately.

Autopsy showed recent hemorrhage on right cortex and chronic parenchymatous nephritis.

The following two cases should not be considered as being epileptic:

CASE II.—Minnie Q., admitted to the Craig Colony November 16, 1906; 52 years of age. Mentality good. Paternal aunt died of puerperal mania, otherwise family history as obtained was negative. Patient said to have had symptoms of her disease begin at the age of 20 years. Assigned cause shock at brother's death after patient had just recovered from severe attack of influenza. Attending physician diagnosed her condition as *petit mal* epileptic seizures, although elsewhere in her application paper he stated that there was



no loss of consciousness. Seizures both nocturnal and diurnal. On patient's admission to the Craig Colony it was noted that her seizures were apparently not of an epileptic type. She complained of having periods of trembling in 1904; these increased in frequency and severity. She had, in 1905, what the physician called neurasthenia. In September, 1905, she had a cramping in all parts except the head. She describes her condition as a "loss of control over herself." In April, 1906, she alleges she had some convulsive movements of the hands, but cannot tell which hand was first affected, although she thinks it may have been the right. Shortly after that she began to have similar contractions in the muscles of the trunk and other extremities so that, as she described it, she became "curled up." Claims she has never been unconscious during these periods. Has at times been unable to walk, but this condition has become less marked, so she can get around with some assistance. Examination at the time of her admission showed that she, when excited, had a chattering of the teeth, and on attempting to walk would fall unless supported, although there was no actual paralysis and on examination nothing abnormal was found in regard to the cutaneous sensibility. Reflexes were normal. After having been under observation for a time, she was treated with static electricity, baths, and suggestion, following which her condition improved markedly, so that she has had no difficulty whatsoever in getting about during the past four years. In this case there is no question but that the diagnosis of *petit mal* seizures was an incorrect one, her condition being entirely that of a hysterical nature.

CASE III.—Agnes M. B. (517); admitted November 16, 1899; age 20 years; student. Negative heredity. Age at onset 19 years. Assigned cause, debility. Probable cause, congenital predisposition. Character of seizures, hysteria (?). Frequency, at first daily; now every two or three days. Both nocturnal and diurnal. Has no aura. Mental state good. Physical condition normal except that reflexes were hypertypical.

History after admission: Patient states that she has never had convulsions, but has "singing" and "screaming" attacks. On night of arrival at Colony had a seizure during which she sang several verses of a familiar song. Was visited by brother December 27, 1899, who stated that she never had anything but "singing" seizures, which are probably not epileptic in character. Showed steady improvement in mental and physical health. Industrious and well disposed; at work daily. During 1900 had 30 seizures, all of above type, and probably not epileptic. In February, 1902, on permission obtained from relatives, patient was operated upon under chloroform anæsthesia, and double ovariectomy performed. Right ovary was cystic, and left much atrophied. Made good recovery from operation. Marked general improvement, and conduct much improved. No seizures of any kind reported since August, 1900. Left the Colony for three months' vacation August 11, 1902, and was discharged November 14, 1902, as recovered, having had no seizures since leaving the Colony.

The next case is one of a type observed not infrequently:

CASE IV.—Albert J. H. (1522); admitted March 30, 1904; age 69 years. Father died at 69 from cancer of the stomach. Mother died at 85 from gastritis; she is said to have been insane for several months at time of patient's birth. One brother died of tuberculosis.

Patient third in family of ten children. Birth natural. Pertussis at 6 years, scarlet fever at 8, measles at 12, diphtheria at 38, the latter said to have been very severe. Typhoid fever at 40 years. No history of any venereal disease. His first convulsion said to have occurred at the age of 64 years. He went home from his work feeling as well as usual, retired and shortly thereafter had a seizure. Second one occurred six weeks later. No assigned cause. Seizures during the next five years averaged one a month, some severe, some mild in type. He is said to have become more irritable and childish. Had as many as six seizures in twenty-four hours. He asserts that he was in good health up to the time of his first seizure. Complains that during the past four years he has had a gradual loss of hearing with a buzzing sound in his ears. Denies ever having received any head injury. Has a double inguinal hernia which he says has existed for 30 years, necessitating his wearing a truss. Was a sailor on the Great Lakes in his youth, since which time he has worked in a starch factory or as a crockery packer. He claims that at the time of the onset of his present condition was in bed for four weeks before he became himself again. During this period he claims he was unconscious. He asserts that his seizures usually occur between the hours of 9 and 11 P.M., just after retiring. No aura. A letter received from his daughter states that six years ago he went to Sacramento, Cal., to benefit his wife's health if possible. While there, his wife died and he was unable to secure a suitable position and, as he had already spent considerable money, he became very much discouraged and nervous. Soon after this he had his first convulsion. She claims he has had periods of absolute forgetfulness during which he had no recollection of his own name or place of residence. Patient is exceedingly nervous. Claims he had bitten his tongue and the inside of his cheeks several times during attacks. He has been told that he gives a loud scream preceding the seizure. During two seizures he has wet his bed. During the past three years the use of his legs has become more and more impaired until now he drags his left foot. Says at times he cannot tell whether he has feet or not. Almost general loss of pain and tactile sense, more marked in lower extremities. Complains at intervals of a feeling of numbness in the radial side of right forearm. Coördinated movements of upper extremities almost normal. No tremors of hands or face. All reflexes diminished. Muscular power in hands below normal. Skin wrinkled, with many folds, with marked senile pigmentary changes. Urine negative. Mitral regurgitant murmur. Well-marked arteriosclerosis. Extremities cold. Pupils equal and react normally. Speech normal. Beginning arcus senilis.

A few days after admission he developed an increased ataxia and incoördination in both legs and some of right arm. Feeling of numbness in both lower extremities. Says he feels as if standing on cotton. Complains of difficulty in urinating, but no trouble with the rectum. Loss of knee-jerks. Loss of pain sense from entire lower extremities except a few isolated areas on inner sides of knees and thighs.

April 12, 1904: Patient now asserts that for a few days before and after seizures he hears voices in the distance calling "Albert," and at such times he has dreams of dead relatives. Becomes confused on day preceding seizures. On examination today the patellar reflexes were well marked. Some swelling and pitting on legs. Conjunctivæ insensitive to pain. Complains of pain in hips and dull, heavy precordial pain. Pain in bones of lower extremities not very

severe. Is unable to discriminate difference in weight of different objects. Few fibrillary twitchings. Says he would rather be dead than continue in his present condition. Orientation normal. Taste impaired and smell absent.

May 4, 1904: Three severe seizures two days ago. Patient is able to walk about the ward and occasionally out-of-doors. Seems to have but little difficulty in walking.

June 22, 1904: Reflexes present, but diminished. Tactile and pain sense present at first test, but after repeated tests no response over trunk. Some result on face, especially left side. An area exists just above iliac crest on left, passing out from vertebral column, also on right, where he has some temperature sense preserved. Later tests show that this zone extends clear around the trunk at the level mentioned. Abdominal and epigastric reflexes both present. When physician stamped on floor patient said he heard no sound. Color sense apparently normal. Conjunctival and corneal reflexes active. Ophthalmoscopic examination reveals no evidence of optic neuritis or atrophy. It was noticed about this time that patient always recognized physicians and nurses, even at some distance. Small doses of bromide, 30 grains mixed bromides, night and morning for a week preceding and a week following the time he expected seizures to occur.

July 9, 1904: No seizures since May 27th. Improved physically. Vision much improved. No œdema of feet. Appetite better.

July 16, 1904: Temperature began to rise yesterday morning until in the evening it had reached 103.4° F. Nothing abnormal to be discovered in chest or abdomen. Throat clear. Mental confusion. No subjective symptoms. This morning temperature, pulse, and respiration are normal.

July 18, 1904: Another rise of temperature in the evening, with fall to normal in morning. Blood examination negative. No chill or sweating. Pulse regular.

August 1, 1904: No seizures since May 27th. Patient dull and apathetic. Inclined to stay in bed most of the time, but is made to sit up daily. Takes nourishment regularly. Now taking 30 grains of mixed bromides daily. Mental confusion almost constantly, although patient does not soil himself.

August 10, 1904: Remains in bed. Subnormal temperature and pulse. Coldness of extremities and marked cyanosis of nails. Continues to take a fair amount of nourishment daily. Bromides stopped. Marked mental apathy. Occasionally soils himself.

August 28, 1904: Gradual failure of condition until death occurred at 3:40 to-day. He continued to take liquid nourishment until the day before his death. Reflexes were present up to 24 hours before death. During this latter period evidences of bronchopneumonia were discovered in both lungs posteriorly. Great emaciation had occurred. Unable to obtain autopsy.

The condition present in this patient was diagnosed at the Colony as hysteria developing on a basis of advanced senile deterioration.

As a point in differential diagnosis it should be remembered that epilepsy is a disorder leading to dementia, while hysteria is not. The perversion of consciousness in hysteria may be confused with or considered as the impairment or loss of consciousness seen in epilepsy,

but ordinarily it does not terminate in mental confusion or automatism. Hysterics recover suddenly from the attack and often express relief after the attack is over. In the hysterical individual there is a possibility of arousing the phenomena by hypnotism, but not so with epileptics.

Oppenheim first described psychasthenic attacks occurring periodically and infrequently in adults who showed a special cause for the attack. The attacks are characterized by prolonged unconsciousness, with perhaps a few muscular twitchings. There is an absence of progressive mental deterioration. A seizure described by Janet, called psycholeptic, presented a feeling of unreality or a dreamy state as the most prominent symptom.

Gowers's vagal or vasovagal attacks and Dana's para-epilepsy are similar conditions. Gowers considers vasovagal seizures as extended epileptic seizures. Russels considers all such seizures, including epileptic, as of vasomotor origin.

Insufficiency of thyroid secretion may produce vasomotor phenomena akin to Gowers's vasovagal attacks or Dana's para-epilepsy. Dana notes an associated thyroidism. Gowers described seizures with definite vasomotor phenomena: throbbing, thumping, and beating of the heart, flushing of face, fulness of the head, dizziness, coldness of the limbs, facial pallor, a shivering perhaps amounting to rigor, tingling and numbness of extremities, and sometimes slight tetanoid spasm. These are considered as closely akin to epilepsy. With the symptoms of the vagal attacks are sensations referred to the stomach, heart, and respiratory organs, nausea, ascent of sensation from stomach to throat and head, apprehension, fear of impending death, and sensations of a vague, indescribable character with no loss of consciousness but a slowing of mental operations, a feeling of unreality, and a great acceleration of the heart's action at the end of the seizure. Seizures consist usually of a sense of apprehension. Most of these symptoms are common as *auræ* in epileptics and in many incomplete or abortive epileptic attacks.

Jones, Fox, and others claim that every symptom of the true *grand mal* seizure may occur in hysteria as well as in true epilepsy. Heilbronner states that epilepsy is not present unless the characteristic mental changes are observed apart from the fits. Some assert that many a supposed typical epileptic attack, especially those of



the so-called psychic nature, prove on investigation to be functional, *i.e.*, hysterical. Sidis says, "The phenomena of 'psychic' epilepsy are of the nature of posthypnotic automatisms."

While Heilbronner will admit no relation between functional and organic symptoms, Binswanger suggests perhaps a quantitative difference in the lesions. The functional neuroses and psychoses have been considered chemiconnutritive, having disturbances of the nerve-cells, "assimilatory" lesions.

Studies of many cases of migraine show that a large proportion of epileptics have a history of having had some migrainous phenomena. Nevertheless, there is no positive evidence that migraine tends to end in epilepsy. Gowers places migraine on the borderland of epilepsy for the following reasons: They sometimes replace one another in the same subject, both have premonitory symptoms, each has a headache as a sequel. He states, however, that there are definite and distinct differences between the two conditions—*e.g.*, duration, character of warning, pain in head.

Periodic headaches occurring in early life may be replaced later by convulsive seizures. When the seizures begin, the headaches may cease or become less frequent. This is particularly true in some cases of senile epilepsy.

Epileptic seizures in early life are occasionally replaced by periodic headaches in after-years. These periodic headaches may be considered as equivalents of epileptic seizures. We must not jump to the conclusion, however, that individuals who are subject to headaches are epileptics. Other symptoms indicative of epilepsy must be present before such a diagnosis can be made.

In the following case it was impossible to definitely establish the occurrence of a true convulsive seizure:

CASE V.—William K. (957); admitted March 8, 1901; age 41 years; railroad man. Heredity: Mother died from tuberculosis; father from "rheumatic fever." Age at onset of epilepsy 35 years. Assigned cause, sunstroke. Character of seizures said to have been *petit mal*. Frequency, once a month, both nocturnal and diurnal. Aura of feeling of coldness. Mental and physical examination on admission normal, except slight mitral presystolic murmur.

After admission patient stated that he had sunstroke in 1894, and was mentally disturbed for eighteen months. Says he has not had a severe seizure for over two years. In excellent physical health; quiet and well behaved. Usually rises in morning with headache. In September, 1902, eyes were examined and glasses fitted. After this headaches became infrequent. Remained at the Colony



in good health until discharged August 16, 1907. Recovered, no seizures having been reported. Readmitted later because of recurrence of headaches, but no seizures observed.

The assigning of various reflex irritations as the cause of convulsive seizures appears to me to be greatly overdone. Many years ago evidence was produced to discredit much of such diagnoses. The hereditary or early acquired defect is overlooked and some condition, abnormal it may be, but not peculiar in any way to epilepsy, is proclaimed as the cause of the convulsions. As a result of such a continued belief on the part of many physicians, we hear of patients in whom intranasal abnormalities, dental anomalies, phimosi, ingrowing nails, worms, hemorrhoids, and a multitude of conditions have produced the symptoms which are truly epileptic and toward which the conditions referred to have but a coincidental relation. I have seen many patients whose disorder was proclaimed as the consequence of a condition which had absolutely no causative relationship, *e.g.*, symptoms of some reflex local irritation, when a superficial examination gave definite evidence of the person being an epileptic, perhaps an infantile cerebral palsy case.

According to Krause, the majority of the cases of jacksonian epilepsy develop on the basis of an infantile cerebral palsy. In these cases occur softening of more or less circumscribed cerebral areas, cyst-formation, atrophic or sclerotic changes, extensive pia-arachnoid œdema, and connective-tissue cicatricial thickening along the vessels. Cerebral palsies of children, especially hemiplegias, may clear up to a marked degree so that in after years they are not recognized unless a careful examination is made, which reveals exaggerated reflexes on the paralyzed side, an occasional convulsion or series of convulsions, following which there may be an exhaustion paralysis, Babinski reflex, and ankle clonus found on the hemiplegic side.

Where criminals are said to be epileptics, it would be well to hold the person under observation sufficiently long to make a positive diagnosis. If it is a true case, it will not be long before some diagnostic symptoms will show themselves—*e.g.*, seizures and mental changes.

The diagnosis of epilepsy is of vast importance in certain occupations—*e.g.*, locomotive engineers, motormen, chauffeurs, etc.—as in these the disease is a serious menace to many.

Many of our cranks and mistaken reformers are abnormal men-

tally and some are without doubt epileptics, displaying frequent psychic equivalents.

In epilepsy we observe certain periodical mental disturbances which appear as transitory—ill humor, dreamy states, stupor, impulsiveness, motor activity, homicidal tendencies, etc. In all these is a more or less marked underlying mental, moral, and emotional weakness with narrowing of the mental horizon and a tendency to circumstantiality in conversation and writing, great prominence of self with impairment of memory, impaired judgment, blunting of the finer sensibilities, and frequently an extraordinary hope of recovery from their disorder. It is observed in institutions that despite these mental abnormalities they are usually attentive to their fellow-patients, assiduously assisting them during seizures. Their capacity for work is, as a rule, materially below the normal for their station in life. The following patient was subject to periods of epileptic stupor, which condition is sometimes incorrectly diagnosed:

CASE VI.—Theodore W. (1427); age 30 years; admitted August 17, 1905, died November 12, 1909. Single, knitter and machinist, also divinity student. Brother epileptic. Father died at 60 years of some paralysis. Mother suffered from periodical headaches.

Patient's first seizure said to have occurred at age of 17 years. Assigned cause, excitement. Second three months later, convulsive in type. Subject to periods of depression. Speech hesitating.

After his admission to the Colony, it was noticed that from time to time he had periods of confusion, one of which was as follows:

June 18, 1906: Eight days ago this patient became confused rather suddenly. He would not answer questions. Remained in bed. Slept well. Remained lying perfectly quiet; when spoken to, gazed at person questioning him, but made no response. Would not show his tongue and resisted opening his mouth. Had to be fed. At one time he made a sign to come to him, but when approached he simply stared and said nothing. One night during this period he arose from his bed and read his Bible. Two days ago he began to clear up and talk, but still seemed rather confused. This morning he seems in his usual condition. He states that the eight days referred to are a complete blank to him and that when he began to recover he could not orientate himself. The last thing he has any recollection of is being in the toilet room eight days previous.

March 27, 1907: Patient is in another period in which he will not speak. He appears dazed, acting almost as if in a dream. When ordered to do so, he walked about and then started to undress. Is in a catatonic-like state. When his arms or hands are placed in a certain position, he keeps them there. If his eyes are closed by the physician, he keeps them closed. If his lids are separated, they remain wide open. Knee-jerks exaggerated on both sides, but no exaggeration of other reflexes.

March 28, 1907: Patient clearer mentally. Says he remembers physician speaking to him yesterday, but felt too weak to answer. Complains of not sleeping well, although nurse reports to the contrary. He says he thought his friends were going away from him on the telephone. Orientation good. Says he has no backache. His tongue is coated and there is some anorexia. No indicanuria. March 29, 1907: Patient is again mute. Shows tongue when asked to do so. No change in his reflexes. Takes food and medicine as given. April 4, 1907: Patient is in his usual condition.

The progressive mental deterioration observed in epileptics is as common a symptom as are the recurring seizures. This is due to the atrophic changes in the essential cells of the cortex, what one would expect in an organic disorder of this nature.

Howden refers to the exaltation of the religious sentiment in epileptics, which seems a strange contradiction to the irritability, suspicion, impulsive violence, and egotism observed in the same class of individuals. He seems to think that perhaps the epileptic child must perforce receive more religious instruction than the average child because of his being kept more at home. Combined with the epileptic's desire for sympathy is a hope of his some time losing his seizures. As Howden expresses it, "This craving for sympathy finds a deep response in the highest development of hope—religion." I will cite some cases to show this emotional, religious trend, in which such messages from the Creator are believed to be received that we can understand how such persons may appear as religious fanatics without their epilepsy being recognized or, if recognized, not appreciated, so far as its bearing on these matters is concerned. It has been claimed that Mother Ann Lee, the founder of the Shakers, who were the former owners of the site of Craig Colony, Swedenborg, Mahomet, and others were epileptic, and that this would explain their alleged visions which brought to them the leadership of religious sects.

CASE VII.—Martin V. (890); admitted January 18, 1901. Father inebriate. Mother insane. Maternal grandmother insane. Patient eldest in family of 6 children. Onset of epilepsy at 3 years. Epigastric aura. Series of 3 or 4 seizures once in two weeks. Physical examination reveals no abnormality. Patient feeble-minded.

Since this patient has been admitted to the institution he has had, at intervals of 3 or 4 months, periods of acute excitement, of a religious nature. In a period which occurred May 1, 1907, he became suddenly disturbed, independent of a seizure, wandering about his cottage, saying, "The house is on fire. I must get out." Shortly after this he was placed in bed and a few moments later was found kneeling by the window, saying, "I see a picture of

FIG. 3.



M. V. Epileptic with marked religious trend.

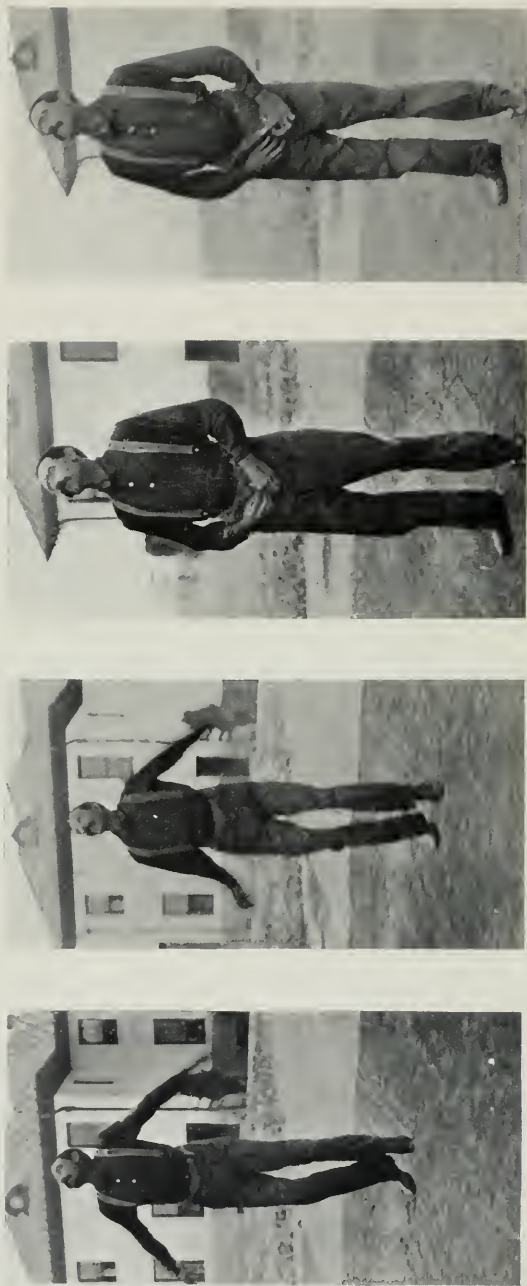


FIG. 4.

E. J. W. Unusual type of petit mal attack, culminating in exhibitionism. Such a patient in a city would be subject to arrest, if seen in the later stage of such an attack.



Saint Ann in the sky. She is going to punish me for the sins of my father." He became much excited when told to go to bed, and, throwing his sheet about him, ran to the roof of the veranda, and, picking up a spindle from a rocker which he found there, he shouted, "I will kill anyone who touches me." After having been brought indoors, he again resumed a kneeling position and said he was praying to Saint Ann and wished to be an example for all young men who did wrong. In another period of excitement which occurred two months later he appeared much confused. He was greatly elated, having an expression of ecstasy. His tongue was coated, conjunctival vessels markedly enlarged, face flushed, exceedingly restless. Was quite well orientated. He talked incoherently as follows: "I have the power in my stomach—in the tip of my tongue. Mr. Peary is looking for the north pole. God Almighty is looking for the north pole. I have the power. I want you to believe me. I have the power."

CASE VIII.—John A. (2374); admitted February 3, 1904; 24 years of age. Father is a clergyman. Patient eldest of 9 children. No history of any nervous disease in family.

Patient's early history negative. When 12 years of age, owing to crowding at his house while his mother was in confinement, he was put to bed with a female nurse who forced him to have sexual relations with her. A short time after he developed evidences of syphilis. Patient was too young to realize what this meant and did not say anything about it until one day his father saw him taking a bath and noticed the eruption. When brought to a physician, it was pronounced syphilis and he was given treatment for three years.

First convulsive seizure at 20 years, but for two years previous he had what he considered to be palpitation of the heart, which he described as a sudden fluttering at the heart with a choking feeling lasting a few minutes. At the time of the first convulsive seizure he ascribed it to the excessive use of tobacco, which he began at the age of 16 years, and says he had a chew in his mouth almost constantly. Second convulsive seizure occurred four hours after first, then none for about 4 months, only he had numerous *petit mal* seizures of the type above described. Preceding his *grand mal* seizures, he claims he had a choking sensation and a feeling of nausea. At times a drink of water will, he claims, ward off a seizure. In the severe seizures, eyes appear to be fixed, patient staring, then he turns to the left, and convulsion follows. A severe headache and drowsiness were present after the seizure, so he was unable to resume work for 24 hours. This headache usually involves the left side. Has become more forgetful, irritable, and fault-finding. Patient was preparing to become a minister, but, owing to his seizures, was forced to drop his studies. Examination of heart reveals no abnormality except a roughening of the second sound. Hands cyanosed and cold. Peripheral circulation poor. Reflexes normal. Slight tremor about mouth and coarse tremor of hands when excited. Mental status good.

After this patient's admission to the Colony it was noticed that from time to time he had periods of mental confusion consisting of marked religious excitement, during which he would also become profane. Sometimes these followed series of seizures during which tongue was bitten. In one of these periods of mental disturbance he said that Christ spoke to him and told him that he would be King of the Jews. "You, John A., will be King of the Jews, a

Saviour of the world. Oh! God Almighty! Thou art in Heaven! You must be Jesus Christ of the whole world. You must be King of the Jews. I am King of the Jews, and put in a rough box. If John D. A. has got that electricity with him, he can be king of the world. Praise His holy name. I am the whole thing. Father A., come here, I am to be king of the nations. I must be the entire saviour of the whole world. I must be the owner of the whole thing. See the pictures up there; aren't they dandy? Oh! God! I will die when they want me. I will show the boys I am clear way ahead. I will take the Calvary and die. I am to be the whole nation's healer. I have got to save this soul of mine. I am to be the nations' healer of the whole University of Jews. John A. is to be King of the Jews. He knows what he had done and he is to be King of the Jews in Jesus Christ's place. Hosanna in the highest and I am King of the Jews. Boys, I have made the whole discovery of the world. Jesus Christ will go to work and let me through." This period lasted for several days, during which time patient's mental condition gradually cleared up.

Masked epilepsy, epilepsia larvata, silent epilepsy, are terms formerly much in vogue to designate patients in whom there was no noticeable motor seizure. It is always questionable as to the advisability of diagnosing the condition unless some, at least, of the convulsive phenomena of epilepsy can be also observed in the same individual.

There is no doubt but that during postparoxysmal periods of mental confusion or automatism certain supposedly criminal acts may be committed—*e.g.*, arson, homicidal attacks, general destructiveness, suicide, indecent exposures, etc.

CASE IX.—Edmund W. (2315); age 36 years; single, farmer. Mental condition good. Is inclined to have periods of depression. Grandfather epileptic. One sister died in infancy, cause unknown. Patient had influenza at 20 years, this being followed by marked prostration, worry, and nervousness. One year later he had measles. First attack occurred at the age of 23 years while in bed. The second one occurred while sitting in a chair. This was two months after the first. His attacks occur at intervals of from a few days to several months. It is claimed that some of his seizures commence with a cramp in his thumb. Patient thought by swinging his hand vigorously he was able to stop some seizures. Seizures were frequent, of the following type: Patient while walking would suddenly take his hat off, throw it in the air, and jump up and down, swinging his arms wildly about. He would then stop suddenly, walk over to where his hat was, put it on his head, and walk off as if nothing had happened. Usually seizure lasted about one minute. When questioned a few minutes later, he would state that he did not know whether he had had a seizure or not and would take off his hat, which might be damaged, and say, "I guess I have."

At another time he was observed to act as follows while having a seizure: While mowing the lawn, he suddenly dropped the handle of the lawn mower, ran

about twenty feet, stopped suddenly, unbuttoned his trousers without exposing himself, and then buttoned up his trousers and walked over to the lawn mower. Physician who saw him at that time stated that his pupils were widely dilated, and that his respirations were short and shallow. Patient said he did not know he had had an attack.

Another time he was lying on the bed when he suddenly jumped up, opened the window, expectorated, then opened his trousers and exposed his genital organs, but did not urinate. When questioned shortly after this, he did not appear to have any recollection of what had occurred.

As Sieveking says, there is scarcely an impression referable to the nerves of common or special sense which cannot appear as a warning of the approach of an epileptic fit, this often being accompanied by a feeling of fear, although occasionally there is a pleasurable sensation. It is well known that these aura sensations are often not correctly recognized.

Mettler says periodical attacks of severe headache, asthma, cardiac disturbances, local and visceral neuralgia, unaccountable moods and spells of eccentricity, vomiting, nocturnal pollution, nightmare, abnormal hunger, and other so-called epileptic equivalents may awaken suspicion of the presence of epilepsy, but, unless a fit is observed, they in themselves could not constitute a basis for the positive diagnosis of epilepsy.

Ambulatory automatism or wandering episodes have been observed in alcoholics, feeble-minded, and demented who were not epileptic. The condition observed in the following two cases is not uncommon:

CASE X.—Bernard H. (2820); age 24 years; admitted July 20, 1909. Father died at 52 years of pulmonary tuberculosis. Mother at 50 of cancer of the liver.

Patient third in line of birth of 6 children. Early history negative. Is said to have suffered from frequent nose-bleeds at the age of 10 years. Began school at 8 years and made good progress. First convulsion said to have occurred at age of 22, at which time he became slightly dazed and staggered, this lasting for about 2 minutes. Second one 7 days later. No assigned cause. Attacks are becoming more severe and are convulsive in nature, patient falling to the ground unconscious, having convulsion and frothing at the mouth, biting tongue and urinating. No warning. Mental condition good.

Shortly after his admission to the Colony he left the institution about 10 A.M. one day without permission. Attendants searched for him for the following 24 hours, at the end of which time he was found at one of the railroad stations on the institution premises. He was confused and could not explain why he went away or where he was during his absence. He did not recognize the physicians or attendants, all of whom were well known to him. He said

his home was in New York City, but for a time he had been at "Craig Cottage." When asked whether this was Craig Colony or not, he was unable to tell. Said he felt very well, that he had eaten only a few apples during the preceding 24 hours. His shoes and trousers were dusty, showing that he had walked some distance in the road. Two days later he was in his usual mental and physical condition, with complete amnesia for the occurrence referred to.

CASE XI.—Robert F. H. (1128); admitted December 14, 1901. Commercial traveller. Father alcoholic. Mother had puerperal insanity after the birth of her youngest child. Both grandfathers were whiskey distillers in Scotland. One brother very nervous. Later it was learned that his paternal grandmother died at the age of 56, assigned cause "softening of the brain." One uncle said to have had general paresis. One aunt was "queer." Paternal grandmother alcoholic. She was thrown out of a carriage on her head and later developed convulsions. She became queer and eventually demented.

Patient states that he was a somnambulist until the age of 12 years. Frequent masturbator between ages of 12 and 22. First seizure said to have occurred at the age of 21, after a hard day's work. This was mild in type, resembling fainting. Second attack 6 months later. Bites tongue occasionally during seizures and has convulsive movements of the upper extremities. No aura. He states that hard work, excess in eating meat, excitement, will bring on an attack. Is frequently automatic following seizures. He claims that he has sensations of a double personality preceding seizures.

This patient gives verified and reliable descriptions of periods of automatism extending over several days, during which he pursued his occupation as a commercial traveller, travelling considerable distances, transacting his business properly and having no recollection thereof; but, from a habit of keeping a diary, could find out later what he had been doing during these periods of amnesia.

It is a common experience to find epileptics, while automatic, to be very resistive and to assault anyone who interferes with them. Such actions may be unrecognized as epileptic and the unfortunate individual be arrested and punished for an assault or even attempt at homicide. A typical automatic state was observed in this case:

CASE XII.—Alfred C. W. (1645); admitted October 12, 1904; age 15 years. Family history negative, except that mother is said to be somewhat nervous. Patient's early history negative, except that his first convulsion occurred when he was two years old; no assigned cause. Second convulsion six months later. At times he had an aura—a feeling of weakness. Some attacks mild, others severe.

This patient on April 20, 1907, complained of a mean feeling in his head. While blacking shoes, he suddenly stopped work, stared fixedly into space before him. Following this there were convulsive movements of the right side of his face with a dilation of both pupils and loss of consciousness, patient falling over on his right elbow but not completely to the floor. Following the clonic movements of the right side of the face, which lasted about 30 seconds, there was a marked pouring of saliva from the right corner of his mouth. No biting



of tongue. No urinating. No stertor. Slight cyanosis for a few seconds. Following the above described symptoms, patient stood up and began to brush his clothing, tried to button parts of his clothing already buttoned, pulled imaginary threads from the air, laughed in a silly manner, looked at observer when the latter spoke to him. Did not appear to comprehend what was said or going on about him. Five minutes later he finished shining of shoes and made change of money offered him for the work. At that time he appeared to be still a little confused.

Certain hysteroid manifestations referred to earlier in this article are seen frequently in the epileptic. Typical examples of this condition are given in the histories of the two cases which follow:

CASE XIII.—Edward J. (1125); admitted December 3, 1901; aged 20 years. Father alcoholic and epileptic. Paternal grandmother epileptic. Mother subject to headaches.

Patient when born is said to have had 4 teeth. Eldest in family of 3. At the age of 16 years, after a three months' debauch and while still intoxicated, is said to have had his first seizure, which was mild in character. Second one year later—*grand mal* in type. Patient states that he began to drink whiskey when 16 years of age and continued practice for 3 or 4 years, being intoxicated on an average of 2 or 3 times a week. Also smoked cigarettes to excess. Has been free from attacks for a period of 3 or 4 months. Seizures said to be nocturnal and occasionally diurnal, and more frequently *petit mal* than *grand mal*. At the age of 10 years patient is said to have fallen from a horse and hurt his head, also fallen from a load of hay and injured his head.

It was noticed after admission that patient had mild convulsive seizures resembling a paramyoclonus. This patient had from time to time for several years an attack of major hysteria, which was promptly relieved by hypodermic injection of one-tenth grain of apomorphine. Patient would sit up in bed, grasp the precordial region with both hands, begin to cry out and moan, and give every evidence of the symptoms of a pseudo agina pectoris. Physical examination gave no evidence of any abnormality of the circulatory organs. Patient's present condition is that of epileptic dementia with recurring *grand mal* seizures of the true epileptic type.

CASE XIV.—Ulysses G. F. (702); admitted April 4, 1900; age 20 years. Father said to have been a moderate drinker. Mother subject to severe attacks of headache. Patient thirteenth child. Labor difficult—instrumental. At the age of 3 years patient said to have had a mild seizure. This type recurred for 2 years, since which time he had both mild and severe. Aura—loud, ticking sound in both ears, followed by feeling of fullness in the head and then unconsciousness. Patient is said to run about before an attack, apparently in fear, having at the time a copious flow of saliva from the mouth.

After his admission to the Colony it was noticed, in addition to typical epileptic attacks, that from time to time he had hysterical seizures, which were characterized by the patient assuming a position of opisthotonos, then continuing a rhythmic, swinging movement of the body, resting on the head and heels. This rising and falling movement would be continued for perhaps half an hour,



or longer if the patient thought he was under observation. Strong pressure on the supra-orbital nerve stopped the movements, which recurred when the pressure ceased. Sometimes accompanying the movement above described, he made a peculiar, grinding sound, synchronous with exhalation. During the intervals between seizures patient frequently complained about gastric distress. At one time he had some mental confusion, lasting over a period of several days. During this he complained of a gripping pain in the abdomen. There was considerable disorientation, with hallucinations of sight. During the latter part of the period of confusion he developed delusions in regard to some unknown person attempting to kill him by cutting his toe-nails. He was very lachrymose at intervals, and, when interrogated, stated he was worrying about his parents, who were dead. A month later he had delusions of persecution, stating that he would kill himself. He stated that some one visited his room at night and put something down his throat; when he tried to remove it, it disappeared. Some one else put hot coals in his bed, causing a hot wave to pass over him.

Certain German observers have claimed that the majority of cases of delirium tremens occur in persons who are epileptic. The patient whose history follows was perhaps of this type:

CASE XV.—James F. (1049); admitted June 4, 1902; 21 years of age, laborer. Elder brother insane. Patient had his first attack at the age of 9 months; assigned cause dentition. Second attack one month later, following which they occurred on an average of once a week and once a month. Severe in type. Has used alcohol and tobacco to excess.

On the day following admission patient had a typical attack of delirium tremens, probably the result of depriving him of the alcohol to which he had become accustomed. A few days later, when he had recovered from this, he stated that he had had delirium tremens on three previous occasions. He informed the physician that he had used beer and whiskey since the age of 12 years, but seldom became intoxicated.

In this case it is a question as to whether the mental disturbance was a true delirium tremens or whether it was a period of epileptic mania, either excited by the abuse of alcohol or independently of the same.

Any convulsive attack occurring after the age of 35 years is, as a rule, due to one or more of the following conditions: Cerebral arteriosclerosis, a syphilitic affection of the cerebral meninges, general paralysis of the insane, intracranial tumor, uræmia, alcoholism, and, in a woman before the menopause, such an attack may be due to eclampsia.

Epilepsy is frequently found in association with other functional or organic disorders of the nervous system. It may so exist with tabes dorsalis, exophthalmic goitre, paralysis agitans, acromegaly, etc.

A patient seen in coma may have sunstroke, cerebral apoplexy,

depressed fracture of the skull, embolism or thrombosis of cerebral vessels, hysteria, diabetes, alcoholic, opium, or other narcotic poisoning, uræmia, or epilepsy, or, what is often forgotten, two or more of these conditions. Many of these states can soon be either excluded or confirmed as a result of the examination and the history obtained from bystanders. These conditions may occur in a person long epileptic, the association being but incidental.

Dipsomania resembles epilepsy in that it is periodical and paroxysmal in type. There are preliminary symptoms of fear, unrest, irritability, impairment of functions of the gastro-intestinal tract. After the debauch there may be delirium or convulsions or both. Dreamy states similar to those seen in epilepsy are sometimes observed.

Acute alcoholic coma does not, as a rule, present convulsions. The odor of the breath, which should not be relied on too much, however; the flushed face, delirium, etc., are not ordinarily seen in the epileptic, although, of course, the two conditions may coexist in the same individual. It has long been known that as a rule an epileptic is very susceptible to even small quantities of alcohol.

A supposed acute alcoholic poisoning may be improperly diagnosed when the pupils are widely dilated, and convulsions occur in an epileptic whose taking of perhaps one small drink may have preceded shortly, or have been coincidental with, the onset of a convulsion.

In uræmic convulsions there may be earlier evidences of a nephritis—œdema of face and other parts, indigestion, urinary changes, vomiting, anæmia, etc., are often present. The finding of albumin and casts in the urine will not permit exclusion of idiopathic epilepsy from uræmia. It is well known that such a finding is very common in epilepsy, especially just after seizures. Furthermore, a chronic interstitial nephritis is common in epilepsy. Uræmia is seldom, and puerperal eclampsia naturally never, seen until an adult age has been reached.

In syphilitic epilepsy we may see partial transient palsies as well as any or every type of seizure observed in idiopathic epilepsy. We have now the Wassermann reaction to aid us in diagnosing syphilis, so are not obliged to rely solely on vague and indefinite histories.

To illustrate conditions seen in this type of patient, the following histories are given :

CASE XVI.—Sarah E. R. (1298); age 46 years, seamstress. Mother's family intermarried in a number of instances. Father alcoholic, dissipated, and excitable; said to have had an uncontrollable temper. Both parents said to have died of dropsy and heart-disease.

Patient second child in line of birth. Mother said to have been broken in health, nervous, and worried at time of pregnancy. Patient had whooping-cough, measles, and scarlet fever, after the last-named developing an "abscess of right ear." At 20 years of age showed a paralysis of the left side, especially the leg. Soon after this the vision in the left eye became poor. Ptosis of left side present. This paralysis was transitory, and patient became better. There is a history of her hair falling out at the age of 14 or 15. Otherwise no accurate data could be obtained in regard to infection of syphilis. (Her admission antedated by several years the discovery of the Wassermann test.) At the age of 43 she had vertigo and nausea, following which a general convulsion appeared. She was told that the right side was affected in the tonic spasm. Other convulsions followed daily. Aura—twitching of right leg and arm, preceded by a sensation of uneasiness. Since the onset of these symptoms the dizziness has increased. Patient is irritable, fault-finding. At the time of her admission it was noted that the reflexes were exaggerated, especially the left plantar, and that she had no disorders of motility except weakness in her right arm.

In March, 1903, it was noted that patient had a tremor of the upper lip and some unsteadiness in gait. Delusions of persecution.

January 1, 1906: Patient is in better general health than she has been since her admission to the institution. More amiable in her disposition.

March 27, 1907: This patient has been mentally disturbed for the past three days, accompanying which there is a slight elevation of temperature. This morning she has pseudomyoclonic movements confined to the biceps of right arm and pronators of right forearm. Physical examination otherwise negative except for heart murmur.

March 30, 1907: Left pupil dilated. Well-marked Babinski on right side. Myoclonic movements continue in right arm. *Grand mal* seizures daily, following which slight exhaustion paralysis on right side is observed. On following day no Babinski could be obtained. Other reflexes apparently normal. Left pupil only slightly larger than right.

Two weeks later it was noted that pseudomyoclonic movements had been absent for several days. Patient continued confused and had a marked paralysis of the right leg, dragging it after her as she walked. Placed on specific treatment. Slight power of movement in fingers and forearm of right side noted one month later.

June 22, 1907: Patient had status period consisting of general convulsions. Recovery uneventful, but remained in bed for several days, complaining of dizziness. Later contractures developed in the right hand.

January 1, 1912: Patient's attacks continue, both diurnal and nocturnal, *grand mal* principally. During 1911 she had 20 *grand mal* and one *petit mal*. Some of her attacks were localized, involving only the paralyzed arm.

This patient had a defective makeup at birth, and in all probability acquired syphilis at an early age. These combined factors resulted in the symptoms which developed later.

CASE XVII.—James H. M. (1507); admitted February 24, 1904; 40 years of

age; white, occupation commercial traveller. Mother epileptic, died during seizure at age of 40 years. Patient third in family of six children. One sister an invalid, others living and well. One aunt neurasthenic the greater portion of her life.

Patient had pertussis at 8, measles at 10, and syphilis at 26, for which he received treatment. On September 8, 1894, while marching in a Knights Templar parade in Buffalo, he fell unconscious and remained so until brought to his home in a distant village. Physicians told him he had a sunstroke. Following this he was in bed for some time and for a long period after arising he had a weakness of his right side, which he claims gradually disappeared, except that his right leg was easily fatigued. Examination showed slight asymmetry of face and the muscles of the left side showed some diminution of power. Marked loss of memory for recent events. Shortly after this he became a bartender, during which time he had a typical *grand mal* seizure. He claims he always had a marked aversion for cigarette smoke, and he believes this seizure was brought on by the smell of this smoke. With this attack, he had an aura of pain in lower abdomen rising to throat, and feeling of weakness. At times he has a burning in his stomach and a feeling that a seizure is approaching, but this will cease if he can procure a glass of water at once. As a rule he had no aura preceding his *grand mal* seizures. Seizures vary in frequency from one, two, or three a week to as many every two weeks. Bites his tongue in severe seizures.

On admission knee-jerks slightly exaggerated; other reflexes normal. Patient well nourished; 5 feet 9 inches tall; weight 200 pounds. Pupils equal and reacted normally to light and accommodation. Speech somewhat slow, but otherwise normal.

Following his admission, he was placed at work with the painter, and later in the garden, but was inclined to shirk.

On September 16, 1904, he eloped from the Colony and, not having returned on November 10, was discharged as improved.

He was admitted to the Willard State Hospital February 3, 1906. About six months previous to his admission to the State Hospital it was stated that he became very violent, turned the family out of the house, threatened suicide and homicide. An abstract of his history while at Willard states that on admission there he gave a quite coherent account of his troubles, thought that cigarette smoke was the cause of his fits; also said that at home and on the trip there was something put on him which was like electricity. He had convulsions fairly frequently and would be dull and stupid after them, for a short time, then irritable, and frequently assaulting. Reacted to hallucinations of hearing, thought members of his family were talking to him. He showed no material change for many months, but then gradually began to deteriorate.

In March, 1909, he had become simple and childish, confused, irritable, and violent after his convulsions.

In March, 1910, he was greatly deteriorated, and was so confused and feeble after a series of convulsions that he had to be kept in the ward for several days. In August, 1910, he had an apoplectic stroke, resulting in a left hemiplegia without complete paralysis. Five days later he had improved slightly, but about three weeks after his apoplectic attack he had a series of convulsions and died from cerebral hemorrhage.



In general paresis and dementia præcox the mental symptoms and history should render it easy to diagnose the condition correctly and not consider the convulsions as those of idiopathic epilepsy. In dementia præcox the onset is at the same early age as is so often seen in epilepsy, the mannerisms, mutism, negativism, stereotypy, less marked mental deterioration in early stages. The previous history of recurring convulsions over a long period, etc., would exclude dementia præcox. In some cases the differential diagnosis is exceedingly difficult. In general paresis there is the onset at later age, the immobile pupils with inequality, the characteristic slurring speech, lymphocytosis of cerebrospinal fluid, rapid dementia, emotional indifference, more sudden change in moral tone, facial tremor, and the history of previous intelligence and usually infection with syphilis, possibly a positive Wassermann reaction.

The following patient is one in whom epilepsy was diagnosed originally, but whose later history leads one to conclude that he perhaps had general paresis:

CASE XVIII.—Mark J. Q. (1225); admitted July 11, 1902; age 41 years; occupation shoe cutter; married.

Onset of alleged epilepsy at 38 years. Maternal grandmother died of paralysis at 60 years of age. Family of nervous temperament. Father alcoholic. Patient troubled with frequent attacks of epistaxis during youth and up to the time of the onset of his epilepsy. He had measles at age of 10 years. At 14 years of age he was struck on the head with a stone. Syphilis denied; wife had five miscarriages, one living child. Otherwise personal history negative. At 38 years of age, while dozing in a chair, after eating a hearty meal, he suddenly awoke and had what he described as a choking sensation. Five days later, while in bed at 6 A.M., he had a similar sensation. These symptoms increased in frequency until they were recurring several times daily. Complained of peculiar odors. Some automatism appeared after these seizures. Later seizures consisted of a loss of consciousness for two or three minutes in which there was a spasm of the right face. Following these seizures he felt dull for an hour or two. Patient never complained of headache. His mental condition failed, so he had to give up work.

After his admission to the Colony he suffered from nostalgia and was very much discontented with his life in the institution. Was placed at various and light occupations, but objected to all work. While at home on a leave of absence in January, 1903, is said to have had a general convulsion.

Continued mental and physical deterioration. Progressive amnesia. The writing was that of a general paretic, as was also the speech. It was noted that patient had all the usual somatic evidences of general paresis, together with a progressive dementia. Occasional brief loss of consciousness with no apparent convulsion, followed by a short period of automatism and mental confusion. Patient had considerable difficulty in feeding himself, soiling his clothing at meals. Had control over the bladder and rectum. Some disorientation present.



January 22, 1904: Speech incoherent, irrelevant, and slurring. Pupils equal and react to light and accommodation. Patient unable to recognize physicians, nurses, or patients who had been closely associated with him for some months past. Dragged right foot. Difficulty in picking objects from floor. When asked days of week, he said, "Andrew." When asked names of month, he wrote figures. Asked where he was, he replied, "Newark, N. J." Then, "I am the coachman of Bismarck. I am in the oil business."

January 25, 1904: Transferred to a State hospital for the insane, where he developed a scanning speech, tremor of tongue. General tremor of body, especially right side, not intentional in type. Reflexes exaggerated generally. Did not appreciate that he was ill and was not greatly concerned about his condition. Convulsive seizures involving especially right side, including face, were observed. These attacks were followed by mental confusion. Right pupil dilated more rapidly than left, and later remained larger, otherwise eyes negative. Temperature sense impaired from right ankle to knee. Posture, pain, stereognostic, localization, tactile senses normal. Biernacki's sign present on right side. Right side face flattened, mouth drawn to left. Weakness right side of body. Movements of tongue normal. No Romberg sign.

November 6, 1905: Patient complained of not feeling well, had severe occipital headache, pronounced tremor of right arm and leg. In morning had convulsive seizure lasting one minute, following which he conversed and complained less of occipital pain. Continues restless, holding right arm and hand with left, moaned and put his hands to his head. At 4 P.M. he had another convulsion, involving principally his right side, following which right side continued spastic, both pupils dilated, left larger than right. Pulse strong and full, rate 80. At 5:15 P.M. another convulsion occurred and patient died. Permission for autopsy could not be obtained. Final diagnosis: Psychosis accompanying organic brain disease.

The diabetic may develop periods of irritability or definite mental disturbance, headache, vertigo, epileptiform seizures, coma with fever, and convulsions. The acetonuria, glycosuria, thirst, polyuria, and emaciation will aid in making the correct diagnosis, although it should be remembered that polyuria is exceedingly common in epilepsy.

An attack of cerebral apoplexy in which convulsions occur may be diagnosed as epilepsy, especially where the seizure is followed by a long comatose stage with perhaps an exhaustion paralysis.

In opium coma the slow respiration and pulse, the pin-point pupils, absence of reflexes, normal or subnormal temperature should exclude a diagnosis of epilepsy, especially status epilepticus.

Convulsive seizures due to lead as the causative factor are oft-times difficult to diagnose, as the obtaining of the history of the contact with lead may be well-nigh impossible.

Syncope is sometimes difficult to distinguish from epilepsy because of the alleged underlying cause, cerebral anæmia, being common

to both. An exciting cause for the syncope, such as a confined room, shock, fright, etc., is usually found on inquiry.

Aural vertigo, or Ménière's disease, is usually accompanied by tinnitus and deafness.

The puerperal eclampsia must be diagnosed from the history of earlier symptoms and the fact that seldom does epilepsy *begin* in pregnancy.

A considerable number of cases are seen in whom arteriosclerosis is apparently one of the essential underlying causative factors. In some persons this condition appears to a marked extent in the third decade of life.

The case cited shows how late in life such phenomena may first manifest themselves:

CASE XIX.—Zebulon W. (1453); admitted October 23, 1903; age 91 years 8 months; a widower. Family history negative. Patient led an active life as a farmer and lawyer until he retired when well along in years. Between the ages of 88 and 89 he found himself on the floor one day and did not know how he got there. Thought that he had fainted as a result of weakness following an influenza. Second seizure several months later, preceding which there was a prolonged cry. He then fell to the floor and had a general convulsion with stertor and coma. No warning. At times he had a twitching of the muscles about the mouth. There is a history of his having fallen from his horse about 7 years previous to the onset of his convulsions, at which time he injured the back of his head. (Examination failed to reveal any external evidence of injury.) Patient inclined to be lachrymose.

Examination at the time of his admission showed a man quite well preserved for his years. Some atheroma of the radial arteries. Heart sounds weak and a mitral murmur present. Double arcus senilis. Says he has an occasional pain in the epigastrium, which is relieved by a drink of hot water or milk. In addition to his convulsive seizures, he has mild seizures consisting of a momentary loss of consciousness. Three times during the past year his bed was found wet in the morning and patient was confused, as if a nocturnal seizure had occurred. Sometimes for a period of 24 hours preceding a seizure patient becomes lachrymose.

After his admission to the Colony he was able to be up and about and walk about the ground for some time.

January 8, 1903: Mild mental confusion present, during which he wandered about his room. Forty-eight hours after this he returned to his usual condition. March 14, heart irregular and about 54 beats to the minute. Complaining of a diarrhœa and neuralgic pain in extremities. March 23: Much improved. About to be up and about the hospital wards. Heart is now regular and diarrhœa has ceased. About 4:30 A.M. the nurse gave him his medicine as ordered and he seemed to be in his usual condition. A few minutes later, when she returned to the room, he was dead. No evidence of a convulsion.

Permission was given for a partial autopsy, which showed heart enlarged, thickening of both mitral and aortic valves. Well-marked arteriosclerotic changes.

Camp refers to seven cases of epilepsy showing normal visual fields in form and color, and seven other cases with a questionable diagnosis of epilepsy showing changes in the color field; one with interlacing fields in both eyes had a history of lead colic and otitis media, and the attacks were atypical besides being hysterical in type. Three other cases of epilepsy showed interlacing color fields, also some hysterical stigmata. Dyschromatopsia is, according to him, rarely if ever present in common epilepsy, but is seen in epilepsy due to brain disease (*sic*), trauma to head or with hysteroid seizures. Tests for dyschromatopsia require intelligent coöperation of the patient, therefore cannot be carried out properly in the majority of epileptics.

Numerous eye conditions are seen in idiopathic epileptics, but it is generally held that these are purely incidental.

C. K. Mills warns the diagnostician to bear in mind (1) that tumors situated in other parts of the brain than the motor cortex may cause jacksonian epilepsy; (2) that other lesions besides tumors situated in the motor cortex may cause this form of spasm; (3) that it may occur in toxic and other diseases in which no demonstrable focal lesions are present; (4) that a spasm closely counterparting the jacksonian type may be observed as a reflex or a hysterical disorder; and (5) that jacksonian epilepsy may be simply an integral part of the entire expression of a case of idiopathic epilepsy.

The cerebral cortex is accepted generally as the part of the localization of the epileptic phenomena, but many authorities tell us that the infracortical centres reinforce the cortex. It should be remembered that tumors of cerebellum may produce jacksonian seizures.

In brain-tumor cases one seeks for changes in the optic discs; local intense headache; focal convulsions which later may become general; vertigo; vomiting, projectile or otherwise; momentary losses or impairment of consciousness; these symptoms very naturally depending on the amount of and parts of brains involved. Remissions, often of long duration, may occur, especially after operative interference.

Two cases of brain tumor are cited, one not diagnosed until autopsy, the other not until his admission to the Craig Colony:

CASE XX.—John S. (1140); admitted January 10, 1902; died May 5, 1903; age 28 years. Farmer by occupation. Heredity as given negative, except that father was alcoholic. Patient fourth in family of eight children. His first attack said to have occurred at the age of 26 years, just after eating dinner and while walking to the barn. This resembled a syncopal attack. Second attack occurred three months later. Psychic seizures during which the patient became violent were also reported previous to admission. It was noticed that the use of alcohol and tobacco increased the number of seizures and that patient was growing more irritable. Patient was accustomed to drink ale from childhood and became intoxicated three or four times a year, after reaching adult life. At the age of 24 he worked for one year as a bartender. Seizures were at first all mild, as above noted, but later became more severe and more frequent, both diurnal and nocturnal. An epigastric aura was present, also queer head feelings, as patient described it. It was noticed that there was a palsy, involving the right arm and forearm, this being first observed about September 16, 1901.

At the time of his admission to the Colony physical examination showed a weak pulse, no heart murmurs, poor peripheral circulation. Fairly well nourished. No abnormality of speech, hearing, or vision. Tongue slightly coated; bowels constipated. Urine normal. Both knee-jerks exaggerated. No disorder of motility.

From March 10, 1902, to June 2, 1902, patient was quarantined because of diphtheria. After release from quarantine he worked on the farm, but complained constantly of headache and failure of vision. On September 1st his eyes were examined and glasses fitted. No apparent evidence obtained during this examination of a brain tumor. December 1, 1902: Patient has averaged 10 seizures, some mild and some severe in type, during the past three months. January 1, 1903: Less complaint of headache. Working out of doors nearly every day. February 1, 1903: Still complaining of headache. Has been given iodides for some time past without any beneficial results. April 1, 1903: Since last note patient has been confused much of the time, making many complaints of a hysterical nature. Is very lachrymose, at intervals complaining of impairment of vision. Twenty-four seizures during past month. May 5, 1903: Patient became restless, excited, and moved constantly about. Had one seizure during the morning. At 10:55 P.M. he was found dead, lying on his left side. No seizure had been observed for some hours previous to this time.

Autopsy showed a rather hard mass about the size of an apple, with an irregular, rugged surface, reminding one much of a conglomerate stone, the irregularity being due to a great number of confluent small tumors. On section the tumor had a mosaic-like appearance, transversed by reddish-brown lines which divided it into lobules of acini. Tumor occupied almost entire anterior half of the left temporal lobe. Microscopical examination showed it to be a spindle cell sarcoma.

CASE XXI.—Frank P. J. (1535); admitted June 1, 1904; born May 12, 1867; married; jeweller by occupation. One uncle said to have had epilepsy.



Father died at 62 years of cancer of the stomach. Mother died at 60 of Bright's disease. Otherwise family history negative.

Patient youngest of four children. Patient himself is married and has three children said to be healthy. Early history negative, except that he is said to have always had a weak digestion and periods of epistaxis. Negative history as to convulsions during infancy. Scarlet fever at 4 years, measles at an unknown age, rheumatism at 17. Common school education, learning easily. Claims he had always been healthy before his present trouble began. No history of trauma; not an alcoholic; smokes moderately. Denies venereal infection. Learned jeweller's trade at 20 years of age, and was in business as a jeweller for 10 years. Then managed a store for  $2\frac{1}{2}$  years. Later returned to his jeweller's business for  $1\frac{1}{2}$  years, when his general health failed to such an extent that he had to give up all work. He also states that he was a bandmaster for many years. His first seizure occurred at the age of 32 years. It was a nocturnal convulsion lasting about an hour. No apparent cause discovered at the time. Second seizure 6 months later. For two years preceding his admission to the institution he had severe convulsive attacks every week or two and mild seizures daily. He described a warning, consisting of numbness and tingling in his right hand. Claimed his head always turned to the right and he then became unconscious. Dazed for some time after seizures. Bit his tongue and wet himself during attacks. At one time he weighed 210 pounds. When admitted to the institution he weighed 160 pounds.

Initial examination showed a fairly well nourished male. Hair thin; face asymmetrical, right side being considerably flattened. Pupils normal except for a slow reaction to light. Vision markedly impaired in right eye, unable to count fingers or distinguish any object. With the left eye he could count fingers a short distance from the eye, but beyond that could not recognize objects. Movements of eyeballs impaired. Vision has been gradually failing for the past five years, more rapid at times, but never periods during which it improved. No apparent impairment of hearing, taste, or smell. Some amnesic aphasia. Chest and abdomen normal. No impairment of swallowing. Slight vertigo and headache at times: otherwise general sense of well-being except at the time of his seizures. No severe or localized headache, backache, girdle, or neuralgic pains. No bone pains. Tactile, pain, temperature, localization, muscle, and stereognostic senses normal throughout. No dermatographia or localized perspiration. Plantar reflexes normal. Cremasteric slow; abdominal, epigastric, and corneal active. Knee-jerk normal on left, exaggerated on right. Wrist-jerks normal. Fairly well marked paresis of right side of face. Patient can whistle and protrude tongue well and move tongue to either side. Muscles of right thigh and calf flaccid. Right leg much smaller than left. No difference in measurements of arms. Patient says this difference in measurement of legs has existed for 25 years. Anæsthesia of left ulnar nerve, right normal. No Romberg symptom. Can dress and undress and feed himself. Disorientation at times because of poor vision. No tremors or twitchings. Recognizes words when spoken, but cannot distinguish the words he wishes to use to express himself. When talked to for a short time he becomes restless, snaps his fingers, throws his hands and arms about and says he is "rattled." Worries a good deal about his condition. At times during the day he says every-



thing before his right eye appears red and then green. His wife stated that at home he was irritable and often depressed. Ophthalmoscopic examination showed atrophy of both optic nerves.

June 21, 1904: Early this morning patient had severe headache, localized at vortex on both sides of median line. This continued about half an hour. Pain was so severe that the patient became pale and perspired freely.

June 24: Lateral and rotary nystagmus is appearing. Patient is complaining of a moderately severe frontal headache.

July 7: Patient spends considerable time lying down. His headaches are now occurring spasmodically and so severe that they cause him to cry out and throw himself about in bed. Some vomiting, but not projectile. Patient is given iodides. Electric tests applied to both sides of body, faradic current, show no difference.

July 23: Right pupil slightly larger than left, both responding to light. Some pain over left eye. Continued blepharospasm on left side.

August 2: Complains of headache and of a numbness of left cheek, nose, left side of forehead, and in the scalp. Some swaying in walking. Takes a good amount of nourishment. Writing fair. Slight deviation of uvula to left. Movements of soft palate normal in phonation.

August 11: Smell impaired, almost totally absent on left side. When he hears a watch ticking or a bunch of keys dropped on the floor, cannot name them, but when placed in his hand he can. Mental processes are all much impaired. Taste impaired on anterior portion of tongue, but not posteriorly. When physician snapped his fingers, patient could not name action, but could imitate it. Not soiling himself.

August 26: Increased difficulty in walking, favoring right side very much. Some tenderness on left side of head on percussion. Patient receiving mercurial inunction.

September 13: Patient is quite depressed. Goes out of doors daily when the weather permits. Had a seizure to-day, the exact character of which could not be ascertained, as no competent observer was present. When seen a few minutes later he showed a marked aphasia, dragging of the right leg, exaggerated right knee-jerk, left absent. No apparent difference in grip of his hands. When told to walk, grasp hands, show tongue, etc., he did so promptly. During the month of September he had three severe attacks.

November 1: During past month has had but one severe seizure. Vision has become so impaired that he can only tell daylight from dark. Continues depressed. Complains of quite constant headache on the left side, not worse at night. Appetite good, no vomiting. Complains of numbness of left side of face, also of right side of entire body. Mercurial inunctions discontinued recently because of his gums becoming tender. Mild seizure noted at this time as follows: Patient standing at time, showed no unsteadiness, pupils dilated. Then became much confused, unable to express himself.

January 20, 1905: Patient recently had three *grand mal* seizures, following which the paralysis of his right side has considerably increased, until he now walks with great difficulty. Lost the use of his right hand, being unable to even raise it to his head. Muscles of the palm, interossei, etc., show considerable atrophy, and the skin has a glazed appearance. Considerable atrophy and at times a marked tremor of the left side of his face. Eyes have a staring ap-

pearance. Unable to turn eyes to right much beyond the median line. Unable to converge. Hearing does not seem involved. He now complains of constant headache of a dull character over the left parietal area with no distinct tenderness. Sleeps well and takes considerable nourishment. Urinalysis negative.

January 30: Difficulty in enunciation. Stereognostic sense somewhat disturbed. Some spasticity in right arm. No marked disturbance of posture sense. Marked Babinski reflex on both sides. Faradic current shows diminution of reactions on right face.

February 5: Some haziness of left cornea. Power of right leg decreasing and atrophy of muscles progressive. Can walk only with great difficulty.

February 6: Large bone flap made on left side of skull and brain exposed, but no evidence of tumor or other abnormality could be discovered. Following operation, patient became conscious in about two hours. Unable to speak. Much oozing from scalp wound.

February 11: Patient has been unable to talk since the operation, but apparently understands what is said to him. Wound dressed under ether anæsthesia and found a marked hernia of the brain with breaking down of the dura and some necrosis of the brain substance. Pulse 130, temperature 99, respiration 16.

February 17: Head has been dressed each morning during past week, but at each time portions of the brain tissue were sloughing off. Patient continues conscious and understands what is said to him, but is unable to talk. Paralysis of right side of body complete, total absence of reflexes.

February 18: Last evening patient's temperature rose to 104, condition failed, and death occurred at 8:45 P.M. to-day.

At autopsy, on removing scalp, a large hernia cerebri was seen, the size of a fist, the brain mass having a reddish appearance. On removing dura it was found that the tip of the left temporal lobe was adherent to the base. On removing the brain it was found that left temporal lobe was larger than right. On making section of the left temporal lobe it was found that it consisted of a tumor mass, gelatinous in appearance. The base of the posterior part of the frontal lobe showed increased consistency, and in cutting into it the tumor was found to continue into the frontal lobe, extending to within  $1\frac{1}{2}$  inches of the tip of that lobe. The tumor was apparently not everywhere continuous, but seemed to consist of several foci, one of which involved the caudate nucleus. In the region of the lenticular nucleus was a hemorrhagic softening focus. The infundibulum was thickened. Tumor was sarcomatous in nature.

In brain abscess we may have epilepsy diagnosed because of the symptoms of vertigo, headache, hebétude, indolence, convulsions (especially in children), episodic attacks of restlessness, confusion or excitement, nonfebrile course of many cases.

The following case is of interest:

CASE XXII.—Grover C. W. (1385); admitted May 9, 1903; age 15 years. Mother died of puerperal fever 11 days after birth of patient. She was said to have been subject to sick headaches once a month. Paternal grandfather died

of some paralysis, the exact nature of which could not be ascertained. Onset of symptoms in patient said to have been *petit mal* and occurred at 3½ years of age. Patient said to have had influenza when about 8 years of age, and after that had a discharge from his left ear. First convulsive seizure said to have occurred at the age of 15 years, severe in character, the left side being most affected. This seizure is said to have occurred immediately before an operation performed by Dr. Frederick Whiting, New York City, for brain abscess of the temporosphenoidal lobe and sinus thrombosis in the descending portion of the sigmoid sinus, at which operation a large clot was removed from the sinus and at least 4 ounces of pus from the temporosphenoidal lobe. He had almost complete sensory aphasia and a loss of power of the brachial group following the operation.

At the time of his admission to the Colony the patient stated that before his seizures the discharge from the left ear ceased, and then after the attack the flow recurred. He complained of frequent general headaches. Vision in his left eye said to be much impaired. Considerable tenderness over the site of the operation. Patient left-handed.

June 20, 1903: A pronounced swelling was noticed along the cicatrix of the portion above referred to, so that considerable bulging was present. This was incised and several drachms of thin pus mixed with blood drawn.

July 31: This patient had a series of hysterical seizures, which stopped when he found that there were no spectators observing him.

May 14, 1908: Patient had a period of mental disturbance during which he held imaginary conversations aloud with God and the devil. Seizures recurring daily, severe in type.

January 19, 1909: Still has discharge from left ear. The following is a description of a seizure observed at about this time: Pupils became widely dilated. There was first a contraction of the superior recti muscles, the eyeballs being turned upward so that only the sclera could be seen. Pupils then contracted and remained so during the rest of the seizure. The head and neck were turned to the left. Legs flexed on thigh and thigh on abdomen. Tonic spasm lasted about 10 seconds, clonic stage about the same time, during which he became violent, trying to bite his hand. As soon as the seizure was over, pupils dilated widely. During the seizure head and neck were strongly suffused. Apparently returned to consciousness two minutes after attack was over, sat up in bed and asked why he was kept there.

January 1, 1911: Patient still has some discharge from the left ear. Subject to mental depression.

Another condition in which syncopal, apoplecticiform, or epileptiform attacks occur is Stokes-Adams disease, a disorder, according to Osler, associated with either derangement of the junctional system of the heart or disease of the nerve-centres of the vagi or of the nerves themselves. This condition has been reported at all ages, but in the majority of cases occurs in men over 50 years of age. Transient vertigo, fainting, or a more or less complete loss of consciousness, with or without convulsions, are symptoms which may result in the

making of a diagnosis of epilepsy. The slow pulse, 40 and under, for long periods, the usual age, the prompt recovery from the attack, may aid in making a differential diagnosis from epilepsy. In some cases reported it would seem almost impossible to absolutely exclude epilepsy.

Myoclonus epilepsy is occasionally observed and may cause some confusion in the making of a definite diagnosis. The following two cases have been hitherto unreported:

CASE XXIII.—Samuel R. (1786); patient is first in family of nine children, two of whom are dead. Brother, Mendel, is a patient at the Colony. Father living, aged 36 years. Mother living, aged 34 years. Grandparents said to be living, of good habits.

Born November, 1891, at full term; labor normal in duration, delivery natural. Weight at birth 6½ pounds. Strong baby, nursed by mother. No convulsions or paralysis immediately after birth. Dentition began at 9 months and was difficult. Began to walk at 10 months, without difficulty. Measles at one year. At 9 months patient is said to have had his first epileptic seizure, lasting ten minutes. Second attack occurred six weeks after the first.

At his first admission seizures said to have occurred once in three months during the preceding three years. "The patient complains of his head, then falls down, the convulsion lasting about three minutes. Foams at the mouth, his hands are closed tightly, eyeballs roll backward. Does not sleep after the attack." Preceding the attack, patient said to have a severe occipital headache. At times patient has what is described as an attack of cramps in his right arm, at which time he runs to his mother, who holds his arm for about five minutes, after which attack is over. During these attacks patient does not fall. Right arm is said to be first and most often affected in all seizures. No paralysis has followed seizures or loss of speech. Has internal strabismus of right eye.

Physical examination: Poorly developed, rather small for age. Height 4 feet 2¼ inches, weight 58½ pounds. No unusual malformation or stigmata. No evidence of syphilis. (This antedated the period of the Wassermann test.) Has a slight rotary nystagmus, more marked when turned to extreme right or left. Hearing defective in left ear. Heart and lungs normal. No apparent abnormality of digestive or abdominal organs. During examination patient had a slight jerking movement of head and arms, sometimes shoulder, these movements being frequently repeated and bilateral.

Reflexes: Plantar, slight, of toe-flexion type. Abdominal and epigastric, not obtained. Deep reflexes all slight.

Marked dorsal curvature of spine. Scapulæ seem to project abnormally at lower corners. Motor function apparently normal, except for the interference with movement resulting from the jerkings above mentioned. These are increased when he attempts to do anything. Mental condition: feeble-minded.

After admission it was observed that the choreiform-like movements of head and arms and rotary nystagmus continue at frequent intervals, especially in the morning. Attended Sloyd School, where he made fair progress. Took



mixture of bromides and magnesium sulphate—7½ grains of former and 11 grains of latter, three times daily.

Removed from the Colony by relatives. During the year 1906 26 *grand mal* seizures were recorded.

Readmitted November 17, 1910. At the time of his readmission it was stated that his parents were first cousins, being Austrian Jews. Personal history states that patient was a blue baby. Otherwise early infancy negative. Contrary to information given at the time of first admission, he is said to have begun to walk at 18 months and talk at 2 years. Said to have been weak in both legs. Attended school at age of 6 years and made fair progress. When about a year old patient fell backward and hurt his head.

At time of first convulsion, at age of 9 months, he is said to have fainted and become blue. Admission paper states that patient does not become as blue as formerly.

This patient and his brother, Mendel, have periods of from several minutes to hours and days during which almost continuous myoclonic movements involving all portions of the body are present. At such times the patient cannot stand, and sits in a chair with difficulty. Movements are so extreme at times as to almost throw the patient from the bed. He has been observed to pass several hours, and even days, practically free from any of these movements. At times patient has been thrown on the floor by the severe involuntary muscular contraction of the lower extremities and trunk. Gait is of about normal type during the intermissions of myoclonic movements. Speech slow and hesitating. Patient has had but meagre education and can read and write with difficulty.

CASE XXIV.—Mendel R. (1785); Hebrew; age 9 years. Patient third child in family of nine children. Brother of S. R. Family history given under the brother's case.

Personal History: No ascertainable prenatal conditions. Born full term. labor normal, delivery not instrumental. Weighed 7 pounds at birth. No convulsions, prolonged fits of crying, or paralysis during infancy. Nursed by mother. Teething commenced at 10 months and not difficult. Night terrors in infancy. Commenced to walk at 2 years, with difficulty. No history of rickets. Received no serious injury during infancy or early childhood. Had measles at 4 years, scarlet fever at 6 years—both mild.

His first attack is said to have occurred when he was 13 months old. During the night he was suddenly seized with severe convulsions, lasting about an hour, after which he fell asleep. In about half an hour the convulsions recurred and he was found to have a marked elevation of temperature, which continued several days. No assigned cause. Second attack occurred six weeks following the period above described. At the onset of seizures patient is said to feel weak in his legs, after which the convulsions occur, the patient falling to the ground. Foams at the mouth, closes his fist tightly, and then sleeps after convulsive movements have ceased. Has warning, but cannot describe.

Examination: Patient appears healthy, but is rather thin and small for his age. Muscles are rather poorly developed. Height 3 feet 10½ inches, weight 45 pounds. No unusual malformations. Cervical glands enlarged. Thyroid not enlarged. Has frequent jerkings of his eyelids. No definite nystagmus. Has a marked internal strabismus of the right eye. Pupils equal, regular and



react readily to light and accommodation. Chest rather pigeon-shaped with fair expansion. Abdominal organs negative. Cutaneous sensibility apparently normal. No subjective complaints. Plantar reflex active, of toe-flexion type. Cremasteric, abdominal, and epigastric, active. Deep reflexes could not be elicited. No evidence of any paralysis. Coördination seemed good. Gait and station normal. In addition to the above-mentioned movements of the eyelids, the patient had occasionally, during the examination, slight choreiform-like movements of the arms, but none of the facial muscles observed. No tremors observed. Mental condition: feeble-minded.

After admission patient had frequent mild attacks, characterized by choreiform-like movements of the head and shoulders, occasionally a nystagmus, which was very marked. Placed on a 50 per cent. emulsion of bromipin.

During the next year this patient had 29 *grand mal* seizures, most of which occurred during the latter part of the year. Removed from the Colony by relatives, contrary to our advice.

At the time of second admission it was stated in his application paper that during his first summer he had diarrhoea and attacks of colic. Is said to have had whooping-cough at the age of 5 years. Following an attack of measles is said to have developed stammering. Began school at the age of 6, but made poor progress. During the time he was examined he had marked myoclonic movements.

In some cases of this disorder there is an increasing intensity over a period of days of the myoclonic movements until finally a severe convulsive seizure occurs, following which the movements abate markedly for days or even weeks. In these two brothers the occurrence of the general convulsions seemed to have no such influence. Previous to their admission to the Colony there had been a diagnosis made of the epilepsy, but not of the complication.

In multiple sclerosis epileptiform attacks are comparatively uncommon, whereas in diffuse or pseudosclerosis they are the rule. Several of these latter cases have been under observation at the Craig Colony, some occurring in childhood, others developing in middle life, and diagnosed as paralysis agitans, multiple sclerosis, locomotor ataxia, etc.

A condition diagnosed as epilepsy in a girl admitted to the Craig Colony is undoubtedly an example of family amaurotic idiocy as described by W. Spielmeyer. Our patient has the early onset, rapid mental deterioration, epileptiform seizures, and progressive retinal atrophy, advancing to complete blindness.

CASE XXV.—Frances H. C. (2694); admitted to the Craig Colony February 12, 1909; age 10 years, November 13, 1908. Father living at 36. At 35 years of age he had "rheumatism" lasting for several months. He is subject to sick

headaches and vomits quite frequently, especially if he departs in any way from his regular diet. Mother living, aged 34, apparently in good health. Paternal grandmother died at 62 years from some chronic disease of the liver. Other grandparents living and well. Two maternal aunts and a paternal uncle died from pulmonary tuberculosis. Patient is second of 7 boys and 3 girls. An elder brother 12 years of age, on April 18, 1909, was totally blind and idiotic. Is said to have been normal at birth—a bright, healthy baby, walking and talking at the usual age. At the age of 5 years his vision began to fail and his mentality also. Later his physical condition deteriorated markedly, so that he became very emaciated. He died March 4, 1911, aged nearly 14 years. For several years preceding his death he was up and dressed every day, but his physical condition was very poor. Until a short time previous to his death he was able to feed himself, but not at the table. Later his condition became such that his mother was forced to feed him and, as she described it, care for him like an infant. Frequently cried at night without apparent cause. He had a cough, but never expectorated, apparently swallowing the sputum. About 16 months before he died, early one morning his mother heard a peculiar sound and going to him found that he had his first convulsive attack. From that time up to his death he had several, the exact number of which cannot be ascertained. Just before his death he had a severe attack of chicken-pox, the eruption being very marked, and patient was apparently extremely ill. Shortly preceding his death his extremities became very rigid, apparently some retraction of the neck, and a marked elevation of temperature. The physician who was consulted said he had spinal meningitis and could live but a few days. Mother stated he was conscious and apparently knew when she spoke his name. Much force had to be used to open his mouth. Practically all his hair fell out. About ten days before his death the odor from his breath was very offensive (perhaps gangrene of the lungs was present). Attending physician certified that cause of death was epilepsy. Another brother, 8 years, February 13, 1909, attended school for the year, but made no progress. He was able to read from the board, but had no apparent command of language. Claimed he could not think of words. Could count his fingers, but slowly. He learned to milk and do some farm work. His mother claimed that apparently his sight was dim. Later it was ascertained that this child became blind and was unteachable, so far as school was concerned, but it was said that he was able to assist with the chores about the farm. His mother stated that he had one epileptic seizure August 6, 1911. The next child, now aged 9, is, according to his parents, brighter than most boys of his age. Has reached the fifth grade in school. All the other children are apparently average children in intelligence, three of them going to school. There are two daughters, one of whom is apparently normal. The other, a twin, aged 5 years, was born prematurely. She is small, but apparently healthy.

Patient, Frances H. C., was born at full term, labor normal. Strong baby. Bottle fed. Walked at 14 months, talked at 18 months. No teeth appeared until one year of age. At 4 years had whooping-cough. Entered school at that age and made good progress. Was always a timid, clinging child and would not play with other children at school. Would stand by the window and read as long as she could distinguish a word in the twilight. At 5 years of age her vision failed. She was kept in school, however, as long as she could see, and was then sent to the Batavia State School for the Blind. First epileptic seizure

said to have occurred at age of 8. This was during sleep. She woke up, cried, "I am dying," and had a convulsion lasting several minutes. Assigned cause indigestion. She was cross and peevish for a day or so after. Second seizure a year later while she was ill in bed with measles. Following the second seizure, the attacks recurred at irregular intervals—sometimes twice a week, sometimes twice a day. Apparently severe in character and ordinarily without a warning. Her mental condition gradually and materially weakened as time went on, she becoming irritable and stubborn and at times silly in her manner. In December, 1908, she had scarlet fever, mild in type.

When admitted to the Colony patient was 10 years of age. Fairly well nourished. Thyroid not enlarged. Was almost totally blind. Could only distinguish between light and darkness. Eyes negative to external inspection, except that pupils were dilated. Both pupils responded to light, but sluggishly. No nystagmus or strabismus. Taste not impaired, but smell impaired, especially on left side. No evidence of paralysis. No tremors or incoördination. Speech somewhat hesitating. Mentality fairly good. Ophthalmoscopic examination made April 29, 1909, revealed a moderate paleness of both discs with well-defined narrow ring of black pigment around the entire border of the disc. The vessels crossing the disc appeared somewhat smaller than normal. At this time pupils reacted promptly to light. Although the patient claimed she could see some, proof of this could not be elicited on careful examination, she being unable to distinguish between light and darkness. While questioning patient she appeared to comprehend poorly many small details regarding her condition. Frequently, when starting to answer, she would hesitate, make a crunching sound with her mouth for two or three seconds, and then speak.

February 1, 1912: Patient's mental condition has deteriorated considerably, so that at the present time she requires considerable supervision. Her seizures recur at intervals of from a few days to one or two weeks. There is complete loss of vision. Discs pale, with more pigment about same, possibly hemorrhages.

With a history of three members of this family developing a similar condition, there seems but little question as to the diagnosis—that of family amaurotic idiocy. This condition, previous to the girl's admission, had been considered as possibly juvenile paresis and possibly amaurotic idiocy of the ordinary type, although that was not positively diagnosed.

Simulation may be so well carried out as to escape the detection of all except the most observant. The simulator cannot change his pupils or reflexes, cannot exhibit a rise in temperature, facial cyanosis, an exhaustion paralysis, ecchymoses, or the stupor and prolonged somnolence of the true epileptic seizure. He tends to overdo his part, elaborating symptoms which are not usually particularly prominent, and *vice versa*. Simulation of seizures is ordinarily done to arouse sympathy or to escape punishment or discipline. The *grand mal* or severe seizure is the one usually feigned, and in some instances so successfully that only skilled observers can uncover the imposture. Chewing of soap will produce the froth, and minor

injuries may be self-inflicted just before the attack is simulated. The malingerer's supposed unconsciousness will yield to a deep pressure on the supraorbital nerve, to a touch on the cornea, to the application of a good faradic current, to a threat to give apomorphine, etc.

It is claimed that narcolepsy occurs entirely independently of epilepsy. Bonhoeffer reported a boy having seizures bearing some resemblance to epileptic *petit mal*, but the patient remained conscious and had no subsequent headache or somnolence. They did not cease under bromide medication. I know of two plethoric young women, not epileptic, subject to recurring brief periods of somnolence, whose general health remains unchanged although these periods have been observed for many years.

# Surgery

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## THE "ACUTE ABDOMEN" IN CHILDREN: WITH SPECIAL REFERENCE TO RARER FORMS OF GENERALIZED PERITONITIS, AND ACUTE TUBERCULOUS COMPLICATIONS

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IN a former communication<sup>1</sup> the acute abdomen, with reference to acute intussusception and acute appendicitis, was discussed. There remains, therefore, a rather large variety of conditions giving rise to acute symptoms, which must always be considered, and these are:

1. Pneumococcal Peritonitis.
2. Streptococcal Peritonitis.
3. Leptothrix Peritonitis.
4. Henoch's Purpura.
5. Acute Emergencies arising from Tuberculous Affections of the Mesenteric Glands and Peritoneum.

*Pneumococcal Peritonitis.*—This condition is now more frequently recognized than formerly, owing, in all probability, to the more satisfactory bacteriological examination of abdominal fluids. In a children's hospital 5 to 10 per cent. of all acute abdominal cases are due to this affection. While most observers draw attention to its greater frequency in the female sex, it was my experience to find in 11 consecutive cases, 7 occurring in boys. It is one of the conditions in children which has not only a seasonal variation, appearing to be more common in summer, but it is often present in epidemic form, along with acute lobar pneumonia, and, though a relatively uncommon occurrence, I have seen three cases in two days.

The source of infection is not always easy to determine, and is

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<sup>1</sup> Volume IV of the 20th Series, INTERNATIONAL CLINICS.



still disputed, though in many cases there is a definite pulmonary lesion, but it seems unlikely that any predominance in the female sex is due to infection from the pelvic organs, as the primary lesion in the chest would seem to suggest that secondary peritonitis is a blood-stream or bowel infection. The more thorough examination of abdominal fluids leads to these cases being recognized more frequently than formerly. They may be divided into three groups:

1. Those in which a pulmonary condition is predominant and the peritoneum becomes affected later.

2. Those in which a pulmonary condition is clinically absent and general peritonitis is the only manifestation.

3. Those in which there is pneumococcal septicæmia, usually rapidly fatal.

*Mode of Infection.*—This may be through one of three channels.

1. By spread from the uterus and fallopian tubes.

2. By blood infection or by transmission through the diaphragm.

3. By ingestion and direct transmission through the diaphragm.

It is difficult to believe that infection occurs by way of the fallopian tube. In none of these cases was there evidence of local vaginitis, which one would expect if the infection had spread up from below, as an analogous condition, due to gonococcus, is associated with definite gonococcal vaginitis. Again, this condition is by no means confined to the female sex, as 8 out of the 21 cases occurred in boys. In 6 of these there was a definite lesion in the lungs, so that the genital organs were not the source of infection; and no definite pathological support has been adduced to support this view. Blood infection would appear to be a more probable cause, as considerable clinical and experimental evidence has been brought forward to show the presence of pneumococci in the blood. Thus, Ewing has grown pneumococci from the blood in every case of pneumonia. This fact might at first sight seem to favor the blood infection theory, but if the peritoneum and other serous membranes are liable to blood infection, one would expect it to occur much more frequently in a condition in which the blood always contains pneumococci, so that this observation of Ewing's seems to be rather opposed to a blood infection. Again, if the peritoneum were infected by the blood-stream, one would expect joint and other complications to be more frequent in pneumonia.

A study of the present series of cases seems to me very much more to favor direct peritoneal infection from the intestinal tract.

We have experimental evidence in this direction in that Jensen produced fatal peritonitis in rabbits and mice fed on pneumococci, the intestine being injected and the lymphoid tissue markedly congested. Some of the most distinctive symptoms of the disease are those referable to gastro-enteritis.

In the last 9 in the above series of cases, 8 were girls, whereas, as previously pointed out, the male sex predominated in the first series. Again, of these 9, 7 occurred between the ages of 5 and 7, during the period of second dentition.

*Gastro-Intestinal Symptoms.*—Vomiting was present in all these cases, generally coming on suddenly. In some there was severe retching, simulating acute gastritis. In one case, where this symptom was marked, coming on after eating bananas, the child died in thirty-six hours of pneumococcal septicæmia; pneumococcal peritonitis was present, and the toxæmia was so severe that the patient succumbed before clinical symptoms of peritonitis became obvious. Diarrhœa, said to be a frequent symptom, was severe in 2 cases and slight in 1.

*Pathological Changes in Abdomen.*—The symptoms resemble those of acute general peritonitis following acute appendicitis, and are often indistinguishable therefrom, except that the pain is not infrequently greatest on the left side. Under an anæsthetic the nature of the condition may be suspected in the absence of any swelling or thickening in the appendix region, so that in persons under puberty with general peritonitis, with acute symptoms and marked gastro-intestinal disturbances, the infection in 90 per cent. will probably be pneumococcal if the appendix is excluded. I have had two cases of primary streptococcal peritonitis during the same period as these 20 cases, and one case of mixed coli and *Leptothrix* peritonitis.

On opening the abdomen the diagnosis can be surmised by two characteristics—the entire want of odor in the fluid of the peritoneal cavity and the character of the fluid. The absence of odor is characteristic in all cases, but the appearance of the fluid varies considerably. In the most acute condition, as seen in case of septicæmia with death within forty-eight hours, the fluid is thin and watery, often rather sticky, and microscopically it contains a large number of pneumococci.

Later it becomes straw-green in color, or even brownish and thick, resembling in one case the creamy fluid seen in a pneumococcal empyema. In the earlier stages there are often flakes of lymph in the fluid. The pus, when thick and creamy, is generally very copious, filling the abdomen like a marked ascites. We have had two cases of encysted pneumococcal abscess in which the pus was almost indistinguishable from the tuberculous abscess of *tabes mesenterica*. It is generally due to a mild infection, and the child is emaciated, resembling one suffering from tuberculous peritonitis.

*Pulmonary Affections.*—Pulmonary disease was present in 6 cases, in 4 of which it preceded the peritonitis, and in 2 followed it (*pneumonia and empyema*). Cases associated with lung disease run a favorable course; 5 out of the 6 recovered, and the one that died was an example of septicæmia with pleural and pericardial effusion and incipient pneumonia.

The differentiation between pneumonia and early pneumococcal peritonitis is in some cases not easy, and I am not at all sure that a mild pneumococcal peritonitis is not present in a large percentage of lung affections.

*Prognosis.*—This is grave. Of the 20 cases above recorded 7 died, giving a mortality of 35 per cent. Of the deaths 2 occurred in patients with acute pneumococcal septicæmia; in 4 there was apparently primarily pneumococcal peritonitis, but no definite lung lesion was to be found; one had definite early pneumonia. Of the remaining 13 cases which survived, there was definite pulmonary lesion in 5, so that the presence of pneumonia or pleurisy in these cases would seem to justify a more favorable prognosis.

*Streptococcal Peritonitis.*—This is a rare condition—I have seen but two well-marked cases.

In one case, that of a boy aged 7, a Mongolian idiot, the symptoms were more those of generalized peritonitis of appendix origin. One symptom markedly prominent was diarrhœa, a condition which suggested the probable diagnosis of pneumococcal peritonitis.

On opening the abdomen general congestion of the intestine, and a considerable quantity of clear, serous, somewhat sticky fluid were seen. No gross lesion of viscera was to be made out. This fluid on examination showed a pure culture of *Streptococcus*. The boy made an excellent recovery.

It is interesting to note that in this case a second attack, less acute in character, occurred six months later. The vermiform appendix was removed at the first operation, although it did not exhibit more evidence of disease than the rest of the bowel. There were no other indications of infection throughout the body.

The second case was that of an infant aged 5 weeks with a history of a streptococcal infection of the right upper extremity.

Six days previous to admission to the hospital, œdema appeared in the left lower extremity. On admission the child had all the signs and symptoms of generalized peritonitis. The abdomen was full of thick yellow pus, which gave bacteriologically a pure culture of *Streptococcus*. The case proved fatal.

*Leptothrix Peritonitis*.—It happens not infrequently on making a thorough bacteriological examination of the pus from these cases of generalized peritonitis that a filamentous form of bacillus of the nature of a *Leptothrix* is observed. I saw in consultation about four years ago an infant aged 4 months suffering from generalized peritonitis.

At operation the abdomen contained a large quantity of a serous purulent fluid which on examination showed this to be the predominant organism. It is possible that this may have been a late invasion of a peritonitis of ordinary pyogenic origin, but the duration of symptoms had been for only two days, and the subsequent course of recovery was mild in nature.

I have not as yet seen a pure *Leptothrix* infection of the peritoneal cavity, but I have, however, seen a pure *Leptothrix* meningitis.<sup>2</sup> The patient, a child aged 18 months, subsequently died of pyæmia. The bacteriological report, by Dr. James Ritchie, demonstrated it to be a Gram negative *bacillus mobile*, somewhat resembling the typhoid bacillus in character. It may be associated with other organisms, such as the pneumococcus, but it is of itself capable of producing acute pyæmia, though in most cases the infection is not virulent. Further particulars of this condition will be seen in the paper referred to.<sup>2</sup>

*Acute Abdominal Symptoms in Henoch's Purpura*.—This condition is not infrequently ushered in by obscure abdominal symptoms simulating acute peritonitis or acute intussusception. The author

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<sup>2</sup> Recorded in the *Review of Neurology* (November, 1908).



has seen 3 such cases, all occurring in boys between the ages of 5 and 9. In one case, a boy aged  $8\frac{1}{2}$  years, severe pain was complained of in the abdomen, associated with vomiting. This continued for three days and the patient passed blood and mucus with the stools. The attending doctor sent him to the hospital as a case of acute intussusception. The abdomen was slightly distended, and there was general tenderness all over the lower part of the abdomen.

Laparotomy revealed some blood-stained serum in the peritoneal cavity. There was intense purpuric injection of the large intestine alone, throughout its whole length. Four days later swelling of both knees and ankles developed, with subcutaneous purpura all over the body. He ultimately recovered.

Another case, a boy, was sent to the hospital with acute appendicitis with fever, pain, and marked tenderness in the right iliac fossa. There was leucocytosis of 16,000.

On opening the abdomen, the cæcum and about two inches of the lower end of the ileum were sharply injected with purpuric spots, imparting an intensely purple color to this segment of intestine. The remainder of the bowel was pale, affording a striking contrast to the injected portion, from which it was sharply defined by a distinct line of demarcation. This boy also developed well-marked purpura around the joints and the soles of the feet. A culture was made from the blood by the pathologist (Dr. Shennan), which revealed a pure *Streptococcus* infection. This patient also ultimately recovered.

It is well that this condition should be considered as a possibility in all cases in which the symptoms are obscure, but it can imitate both acute appendicitis and acute intussusception in a very marked degree.

*Acute Emergencies in the Course of Abdominal Tuberculosis.*—Although one must look upon peritoneal and mesenteric glandular tuberculosis as a chronic condition, still throughout the course of the disease, even at its onset, the signs and symptoms become so marked as to simulate many of the acute conditions already mentioned.

A little girl, aged 8, was recently seen in consultation, suffering from acute abdominal symptoms. Some weeks previous she had whooping cough. A week before, she was seized with pain in the abdomen, vomiting and general swelling, and tenderness, which persisted, with a temperature varying between  $101^{\circ}$  and  $103^{\circ}$  F. during that period. On examination there was no special tenderness in the



appendix region. A provisional diagnosis of pneumococcal or acute tuberculous peritonitis was made.

On opening the abdomen a large quantity of serous fluid escaped. The intestines were acutely congested, but on careful examination the bowel and parietal peritoneum were seen to be covered by fine miliary tubercles, detected with difficulty by the naked eye. It was the most acute form of peritonitis of tuberculous origin I have ever seen.

The clinical course was in many respects identical with that of pneumococcal peritonitis. The appearance of the peritoneum pointed to a blood-stream infection. The mesenteric glands were slightly enlarged and in no case showed caseation.

Tuberculous infection of mesenteric glands do not in themselves cause acute symptoms. I have recently had under my care a child aged 6, with an acute abscess of the enteric mesentery, without appendix inflammation, or any other primary source of invasion. Pus showed a pure staphylococcal infection.

The causes of obstruction in tuberculous peritonitis are as follows:

1. Stricture of the ileum most frequently within six inches of the ileocaecal junction.
2. General peritoneal adhesions in a healing peritonitis causing multiple kinking of intestine.
3. Strangulation by a band or adhesion of intestine to a caseous gland, as a rule in the ileocaecal region.

Stricture of the intestine occurs in older children and causes chronic obstruction. It is generally single but occasionally multiple. I have seen one case in a child aged 13 months. This was associated with caecal tuberculosis. Resection of six inches of ileum with caecum and ascending colon resulted in complete cure of the condition. The child is now, six years later, strong and healthy.

Obstruction from general peritoneal adhesions is common in young children. It may follow the healing stage of general miliary tuberculosis or what is sometimes called the fibrous form of tuberculous peritonitis. It occurs in young children more frequently than in older ones.

In the last variety, which is by no means infrequent, the changes in tuberculous mesenteric glands are similar to those in tuberculous glands elsewhere. They are subject to frequently recurring attacks of periadenitis. When one considers the analogous condition in cer-

vical gland tuberculosis, one can readily realize how easily a coil of intestine may become adherent to such a gland. I have seen 4 such cases, in all of which the glands were in the ileocaecal region.

If one examines the anatomical position of these glands, some will be found situated close to the mesenteric attachment of the intestine, and paradenitis and caseation lead to adhesion and dragging on such a coil of intestine, causing kinking, or the formation of a sinus into the intestine, from which a tuberculous abscess forms, which frequently points in the umbilical region.

Many cases of tuberculous umbilical fistula have such an origin, and in operating upon these, a long fistulous track can often be traced passing down to the ileocaecal origin, and having its beginning in the gland which has become adherent to and has burst into the intestine.

These conditions have occurred in my own practice and include the most common causes of the "acute abdomen" in children. More rarely there occur acute inflammation of a Meckel's diverticulum and acute torsion of an ovarian cyst, of which conditions I have had a single instance.

## ADULT FLAT-FOOT

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*Synonyms.*—Pes Valgus, Pes Planus, Weak Foot, Weak Ankles, Everted Foot, Kidney-Foot, Liver-Foot, Splay-Foot, Depressed or Fallen Arches, Low Arches.

*Definition.*—As used by both the general medical profession and the laity, the term "Flat-foot" embraces many more symptoms than would be included in a strict interpretation of the terms "Pes Valgus" or "Pes Planus." It might well be defined as a painful condition of the foot due to abnormal static conditions, the chief of which is "the persistence of the passive attitude." It is characterized by the pronation of the foot upon the ankle, and the breaking of the foot at the medio-tarsal joint with the abduction of its anterior half, together with a displacement of the bones of the ankle and a flattening or lowering of the longitudinal arch of the foot. It is also characterized by the rotation of the legs, the abduction of the feet, and by a marked alteration in the gait.

*Frequency.*—The past ten years have seen a most amazing increase in the number of static foot troubles, so that, outside of acute coryza, rhinitis and gastric disturbances, they constitute the most frequent ailment of the inhabitants of the cities of the United States and Canada. The reason that the physician does not see more of them lies in the fact that the treatment is mostly in the hands of shoe-salesmen and chiropodists. There are scores of factories in America devoted to the manufacture of flat-foot arch supports or braces, for the shoe stores alone; an insignificant one located in the writer's home city produces and sells to shoe stores over 3000 pairs per month, as was lately testified to under oath. This enormous increase is due, without doubt, to the demands of the modern fashions, which to-day call for high-heeled, narrow, and pointed shoes and pumps, together with excessively tight stockings, to be worn from

morning till bedtime for all purposes and occasions. In previous decades, except possibly for use at dancing or at weddings, only the idle rich (then few in number) wore tight shoes with slender high heels—popularly called French heels; but to-day even the school girls of our cities imagine that they would lose caste and be considered “*de trop*” if they could not at least wear high “Cuban” heels and pointed toes all day long. “Common-sense” shoes are no longer to be purchased in the fashionable boot shops nor in those catering to the wants of the laboring people, so that in many instances, especially in small communities, women are compelled to be improperly shod through the utter impossibility of buying correct shoes. The shoe dealer is not to blame, for there seems to be so little demand for the correct shoes that he cannot afford to keep them in stock. This is especially true of the smaller stores and of those in small cities. In the larger cities the females coming for treatment outnumber the males five to one, although the occupations demanding excessive strain upon the feet are held chiefly by men (salespeople in the larger dry goods and department stores excepted); in small communities the affliction is more equally divided between the sexes.

*Causes.*—The REMOTE or underlying causes of static flat-foot may be congenital, neurogenic, inflammatory, traumatic, and constitutional.

1. The foot of the newly-born looks chubby and flat, on account of a cushion of fat which lies beneath the arch and fills out the vault. This is a normal condition, and is always to be differentiated from true congenital pes valgus, which is usually combined with more or less calcaneus, giving the condition best described as congenital pes calcaneo-valgus (Fig. 1).

Due to hereditary influences, the persistence of which is often witnessed in a most striking manner, there may exist at birth, or appear before the child has ever stood upon its feet, a tendency for the foot to pronate and for the child to stand on and walk upon the inner border of the foot (Fig. 2). This is often overlooked, or the condition may remain latent until after puberty, when a more or less severe static flat-foot is discovered. In one family under the writer's observation a father and five sons, as well as all the father's living brothers and sisters to the number of four, have marked static flat-foot. The entire family is of a large, loose-jointed, flabby, fatty,

FIG. 1.



Congenital flat-foot.

FIG. 2.



Pronated foot in a child of 2 years.

FIG. 3.



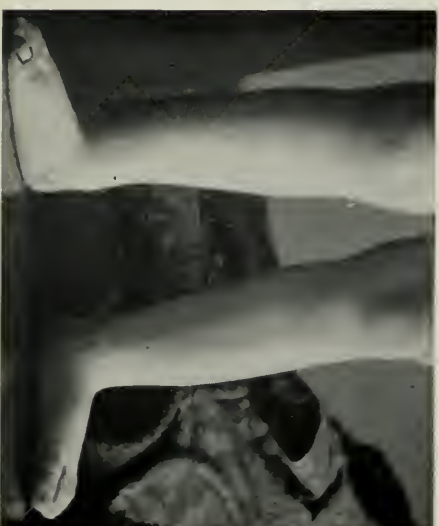
Congenital flat-foot.

FIG. 4.



Pronated foot from contracture of the peronei muscles.

FIG. 5.



Severe type of adult flat-foot, dating from trauma 34 years before.



FIG. 6.



FIG. 7.



Farrier shoeing horse. Toes turn in. Foot adducted.

Farrier's helper filing shoe at bench. Legs in Annandale's position of rest. Note abducted left foot.

awkward type, typical of what the laity term "liver-foot" (Fig. 3).

2. The paralysis or weakening of the supinator group of muscles especially the *tibialis anticus* and *posticus*, leads to the formation of a paralytic flat-foot. Where such paralysis has been recovered from, excessive strain, faulty weight-bearing, or even the unopposed pressure of the bed clothes can in time lead to flat-foot.

Permanent contractures or spasticity of the supinators give rise to most stubborn conditions. Cerebral palsies of various kinds need only start the abnormal process, then faulty weight-bearing and use continue it (Fig. 4).

3. Inflammatory changes in the bones and joints of the tarsus, such as arthritis deformans, gout, acute rheumatism, gonorrhœa, etc., are not infrequent etiological factors. Inflammatory changes in the calf muscles or severe inflammations of the skin and subcutaneous tissues may be the beginning of the subsequent static foot changes.

4. Constitutional diseases affecting the density and strength of the bones must also be considered as contributing factors. Rickets, chondrodystrophia, osteoporosis, osteomalacia (congenital or acquired), give rise to alterations in the structure, shape, and position of the tarsal bones.

5. The most common traumatic cause of flat-foot is Pott's fracture (supra-malleolar fracture), which has not been properly reduced and which has been allowed to heal without the complete restoration of the normal relations between the leg, ankle, and foot. The violence which tends to produce a Pott's fracture is the forcible pronation of the foot, and in the typical deformity the foot and lower fragments stand in abduction and pronation. The joint space between the malleoli is widened, allowing the astragalus to twist or move *in toto* to the outside of the normal position and thus shift the line of gravity inward. Unless special attention is given to the proper adduction and supination of the foot in reduction and fixation, a permanent tendency to the formation of the flat-foot takes place, even though the fragments of the fibula and tibia seem to be in proper position.

Fractures of the *os calcis*, astragalus, or first metatarsal, as well as rupture of the plantar fascia or the lateral ligaments of the ankle, not infrequently lead to altered function and flat-foot (Fig. 5). Trauma to the muscles of the leg can also give rise to similar changes.

The DIRECT cause of all cases of static flat-foot, whether influenced

by any of the above-mentioned primary or contributory causes or not, is the alteration in the static conditions of weight-bearing, posture, or gait, or a change in the balance of the body from such conditions as knock-knee or coxa valgum, bad shoes, etc.

After pregnancy, typhoid fever, surgical operations, lingering diseases, etc., the structures of the feet are often weakened and unable to hold up the weight of the body. A too rapid increase in weight after such conditions, or even in health, may so strain the feet that abnormal function takes place.

Unusual physical exertions in athletes or professional strong men, or the strain of the daily routine in those following occupations in which *standing* rather than *vigorous walking* and *exercise* of the feet are the leading physical exertions, such as policemen, machinists, salespeople, etc., especially when the proper shoes are not worn, tend to destroy the proper balance and function of the feet. The farrier who nails the shoes to the horse's hoof, grasping the hoof between the knees and steadying the body by adducting the feet and rotating them inward (pigeon toe, Fig. 6), never develops a flat-foot, while his helper who stands all day at the forge or vise soon throws the limbs into the "position of rest" and abducts the foot (Fig. 7); it is the helper who is more often afflicted with flat-foot.

The commonest of all causes, however, is the alteration in the shape of the foot, the strength of its muscles and the area of its effective weight-bearing surface due to improper shoes. The normal unclad foot is broad and short, with the measurement across the toes as great as, or greater than, that across the ball of the foot (Fig. 8). This is best illustrated by the feet of the unclothed savage (Fig. 9), which, though they may apparently be flat and even slightly pronated, are held in the strong foot position (Fig. 10) by the broad sweep of the toes, and especially the adduction of the prehensile big toe. The entire tendency of the fashion of all ages and climes has been to compress the toes and ball of the foot into the narrowest possible space, and (in Europe), by raising the heel from the ground, to cause only the compressed toes and ball to touch the ground, thus giving the appearance of a tiny, narrow foot (Cinderella), so sought after by the devotees of Fashion. Such a shoe would to-day be advertised as having "Cuban heel" and a "short vamp" (Figs. 11 and 12). Tight shoes cause foot-damage (Fig. 13).

FIG. 8.



Imprint of soles of Slavonian farmer's wife who rarely wears shoes. Note adduction of great toe.



FIG. 9.



Feet of unclothed savages.

FIG. 10.



Broad and short feet of unclothed savages. Note the prehensile big toe.  
(Figs. 9 and 10 are used through the courtesy of Dr. Phil. Hoffmann, St. Louis.)





X-ray photograph of foot in high-heel shoe. Weight is borne by the ball of the foot.

FIG. 13.



Tight shoes given out by Public Associated Charities.

FIG. 15.



Compression of toes from short tight stockings.

FIG. 16.



Marked flat-foot showing effect of tight shoes upon toes.

FIG. 17.

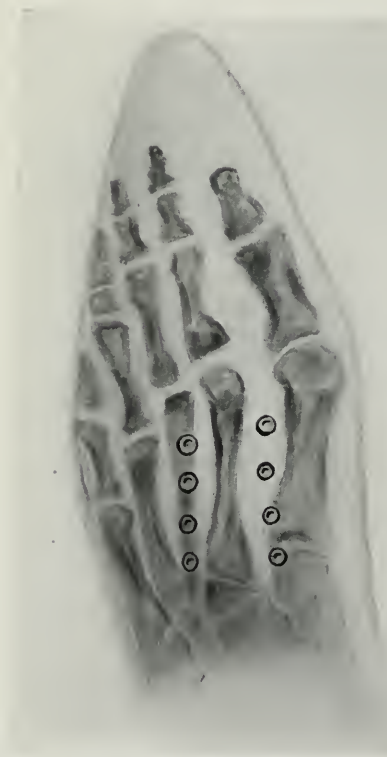


FIG. 18.



Fig. 17.—X-ray of foot pinched in narrow shoe.

Fig. 18.—X-ray of companion foot to Fig. 17, with upper leather cut away, allowing toes to spread.

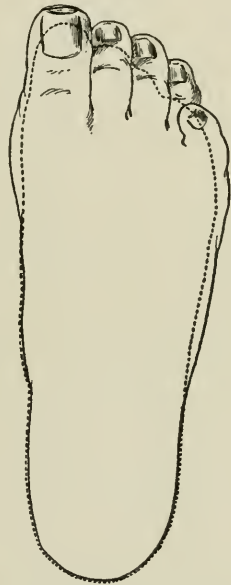
In the United States, in the summer of 1911, Fashion decreed that the shoes have extraordinarily high heels, narrow, pointed soles, and extremely short vamps; they produced an extreme degree of toe walking with lateral and antero-posterior compression of the balls of the feet. The rôle of the stocking in compressing the toes and feet has never been sufficiently emphasized. To produce an illusion of sheerness (thinness of fabric and transparency) it became the practice to knit stockings of especially strong cotton or silk threads,

FIG. 12.



Cuban heel. Gives poor instep support and has all of the disadvantages of the French heel. (Through the courtesy of Dr. A. G. Cook, Hartford, Conn.)

FIG. 14.



Compression of foot from stocking.

with the feet, ankles, and lower leg several sizes too short and narrow, so that when the stocking was stretched out over the foot and ankle, there was produced the maximum opening between the meshes. This could, of course, be accomplished only by the maximum compression of the foot, which in many cases was more severe than that produced by shoes (Figs. 14 and 15).

The compression of the toes and the abduction of the great toe lessen the effective weight-bearing surface of the foot (Figs. 16, 17, 18, 19, and 20) and weaken its ability to adapt itself to the strains

of pronation, and allow permanent twisting to take place more easily. The axes of the feet come to diverge in walking, compelling the patient to roll the weight off the inner border of the foot, thus bringing greater strain upon the inner side, which again favors rotation of the foot and pronation. In this stage the condition is spoken of by Whitman as the "weak foot."

Another factor is that the high heels usually have a very small bearing surface and soon run down on one side, so that the individual

FIG. 19.

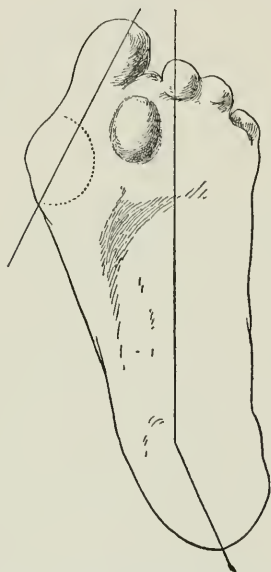


FIG. 20.



Fig. 19.—The effect of short shoes, lessening the weight-bearing surface; the toes do not help bear the weight of the body. Falling of the transverse arch with large callus under the ball of the foot.  
Fig. 20.—Compare tracing of foot to the size of narrow short sole.

is perforce compelled to twist the ankle at each step (Figs. 21 and 22). The stilt-like heel, exerting great leverage, often shifts or bends forward or sideward *in toto* under the weight of the body (Fig. 23). The shoes of to-day are also built to present a peculiar outward sweep of the sole and upper leather for the purpose of showing "artistic lines," so that an experienced observer putting on such a shoe will experience a peculiar tendency, compelling pronation and divergence of the feet.

*Pathology.*—The most important changes from the normal anat-

omy occur in the astragalus, which becomes twisted and articulates more or less faultily with the external and internal malleolus; it becomes flattened and rotates so that the head of the astragalus becomes prominent on the inner border of the foot. The lesser process disappears and there may finally be complete destruction or separation of the cartilage.

The scaphoid becomes displaced laterally and twists upon its sagittal axis so that the tuberosity points downward toward the sole. The change in the relationship between astragalus and scaphoid leads to irritation of the periosteum and osteophytic deposits, which may in turn cause limited motion between these bones.

The os calcis seems to be turned outward; its anterior surface may show two, or even three, articular surfaces; its cartilage may become eroded, and it may finally become ankylosed to the astragalus.

The cuboid is pushed upward and its upper portion becomes prominent; its articular surface is no longer parallel with the os calcis, and on this account the inner edge of the latter may press upon the calcaneo-cuboid ligament.

The plantar fascia becomes relaxed, longer, and thicker. The dorsal ligaments are thin and show degeneration, while the tibialis anticus and posticus muscles are weak and degenerated and their tendons are stretched.

*Morbid Physiology.*—To understand the changes in the position, shape, and function of the feet, a short description of the normal is necessary.

The normal function of the foot is to act as a lever in lifting the weight of the body off the ground in walking. The proper method is for the heel to strike the ground first; immediately thereupon the body goes forward and the heads of the metatarsals (ball of foot) touch the ground and receive the weight, after which the calf muscles contract and raise the body upon the toes, using the tarsus and metatarsals as a lever of the second class. As the foot is plantar flexed upon the ankle to lift the body, the forefoot turns slightly inward. This is best illustrated in athletic sports, in statues of gladiators, discus throwers, etc., and is called the "strong foot" gait (Fig. 24). Normally the axis of the foot (the lever) is held in the sagittal plane of the knee and ankle joints.



When used for passive weight-bearing the legs and feet assume a posture known as "Annandale's attitude of rest," in which the weight of the body is no longer carried by the muscles and joints, but by the bones and ligaments. The legs are slightly separated, the pelvis tilts forward and upward; the hip, knee and ankle joints flex slightly and lock by a slight outward rotation of the legs, and the feet abduct and pronate. All this tends to throw the centre of gravity toward the inner border of the foot, which becomes pronated to reinforce it: if attempts be made to walk in this position, the gait becomes

FIG. 24.

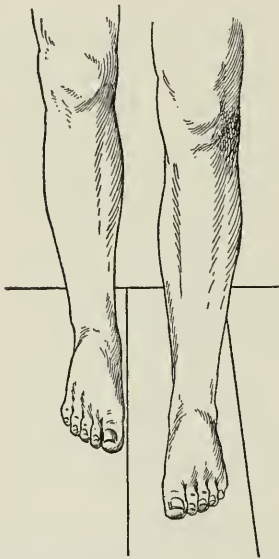


FIG. 25.

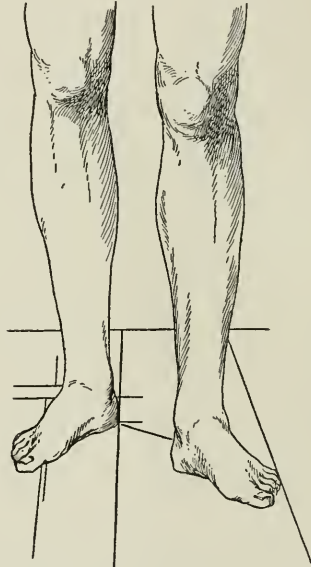


Fig. 24.—Strong-foot gait; feet held parallel.

Fig. 25.—Weak-foot gait; feet turned outward; weight of body rolls off inside of foot. (After Whitman.)

shuffling, and one cannot roll the weight of the body off the balls of the feet, as before described.

In flat-foot this inclination and tendency toward abnormal function has become an accomplished fact and is persisted in habitually. The feet are no longer held parallel, but are more or less widely diverged (Fig. 25); the foot becomes pronated upon the ankle and abducted in the medio-tarsal joint, as though it were broken in two at the scaphoid and the fragments bent outward (Figs. 26, 27, 28, and 29). The great toe becomes abducted and twisted.

FIG. 21.



Eversion of right ankle from twisted high heel.

FIG. 22.



Same patient as in Fig. 21, wearing a low-heel shoe; no eversion.

FIG. 23.



High Cuban heel pushed forward *in toto*.

FIG. 28.



FIG. 29.



Fig. 28.—Severe flat-foot; the foot is broken at the medio-tarsal joint and the halves rotated outward. Note abduction of big toe.  
Fig. 29.—Weak foot; practically no rotation at the medio-tarsal joint. Note marked abduction of big toe.

FIG. 30.



Twisting of the tendo Achillis and os calcis.

In walking, the centre of the heel no longer meets the ground, because the os calcis has become twisted (Fig. 30); the weight of the body does not roll off the toes, as in the strong foot, but is transmitted by means of an outward shuffle of the foot to its inner border and rolls off the inner edge just at the base of the great toe. The gait becomes a shuffle, the leg is rotated outward, and the hips and knees become partially locked; the ankle joint is held stiff, or, rather, does not plantar flex, and the foot performs a peculiar inelastic twist outward as soon as the heel strikes the ground. The shoulders and body sway

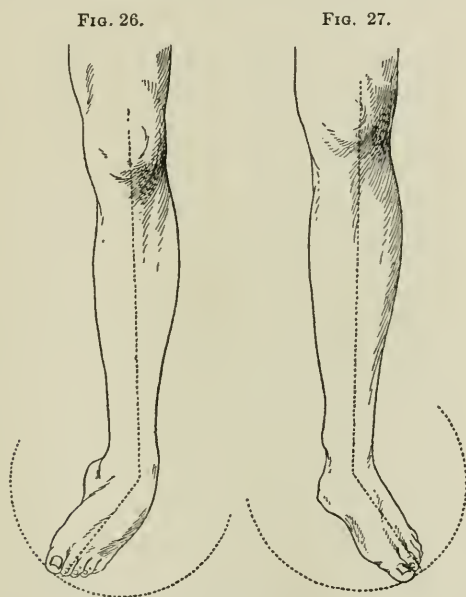


Fig. 26.—Voluntary adduction and supination.  
Fig. 27.—Voluntary abduction and pronation. (After Whitman.)

at each step, and there is a tendency to throw one knee in front of the other as in knock-knee (Fig. 31).

*Symptoms.*—The most common and most important symptom is pain. It is felt most severely when the foot is used, and has no especial localization. It may be felt in the bones of the weakened arch, the dorsum, in the heel, under the malleoli, beneath the tendo Achillis, in the calf, knee, thighs, hip, and even in the sacral and lumbar regions. The onset of the foot-pain is generally quite characteristic. After a night's rest the patients have generally

forgotten the fact that they have feet, but the first few steps out of bed bring back the pain as uncomfortable as ever. In a few moments, after a little brisk movement or a hot bath, the pain disappears, leaving an uncomfortable sense of weakness in the feet, which grows more severe as the patient walks about, until, after several hours' use, the feet begin to hurt and continue to grow more painful as the day passes, unless the feet can be rested. The agony of this continuous pain has, in many cases, caused men and women, otherwise healthy, to give up active life. In other cases it is only the sudden change from sitting or resting to standing or walking, or some violent or unusual motion, such as jumping on or off a street car, or running downstairs, which is painful. Early or milder cases may not suffer actual pain at all. There may be only a sense of weakness, strain, or insecurity about the feet and ankles. "Weak ankles" is the term applied by the laity. "Only after a long time may the patient become aware that he is accommodating his habits to his feet; he rides where once he walked, he sits where once he stood; he complains that he cannot buy comfortable shoes—the shoe that was comfortable in the morning becomes uncomfortable at night—and there is increasing discomfort from corns, bunions, and other preëxisting deformities" (Whitman). The feet may be cold, cyanotic, numb, swollen, and frequently perspiring. The ankle is frequently "turned out"; sprains become common; the muscles of the calf become spastic; the toes may become dorsal or plantar flexed and the ball of the foot (metatarsal heads) painful (Figs. 32, 33 and 34).

The severity of the subjective symptoms, above described, bears absolutely no relation to the severity of the objective symptoms, and *vice versa*. It is a common occurrence to find patients whose sufferings are intense and yet upon careful examination to find few or even no objective symptoms to account for them. The actual symptoms of pain in such cases are due to a disproportion between the burden of the strain and the ability of the foot to sustain it. The pain is most severe when the arch "is falling." It is in these cases that the diagnosis is difficult, and the patients are often dismissed with the recommendation to take a course of baths for rheumatism at some famous bath resort.

*Objective Symptoms.*—The gait of the patient has become slipshod and shuffling: the whole body, especially the shoulders, sways



FIG. 31.



The shuffling gait in flat-foot.

FIG. 32.



FIG. 33.



Fig. 32.—Flat-foot with turned-under toes from use of excessively short shoes.  
Fig. 33.—Severe fixed flat-foot with contracted toes.

FIG. 34.



Weak foot with dorsal contraction of toes

FIG. 35.



Marked flat-foot. Internal malleolus, head of astragalus and scaphoid in straight line.  
Marked sinking of scaphoid.

from side to side as in knock-knee; the feet turn outward and are pronated, so that in walking the patient first strikes his heel and then rolls the weight of the body off the foot at the inner side just above the head of the first metatarsal. Calluses and bunions are common in this particular area. The knees are not fully extended in walking and also never well flexed. Often a slight knock-knee has developed. The shoes are bulged downward at the arch and worn away at the inner side of the sole. The arch of the foot has sunken down, to a greater or lesser degree, from an almost imperceptible amount, to a degree where the arch "digs a hole in the ground." The amount of the surface of the sole bearing against the ground is increased, as can be shown by an imprint, and the range of motion of the foot is decreased in every direction, especially in supination, until in severe cases the foot becomes quite fixed in the abducted and pronated position. There are certain points about the foot which are usually quite tender upon pressure: the head of the astragalus, the scaphoid bone, the dorsum of the foot at its highest point and just below either malleolus. There may also be pain and tenderness beneath the tendo Achillis. The internal malleolus, head of the astragalus, and the scaphoid lie in a straight line and form a convex bulging anterior to and below the malleolus (Fig. 35). It has been suggested by Heines that the United States Navy adopt the arbitrary limit of one-half inch for the amount of depression of the scaphoid allowable as physiologic. Any case in which the scaphoid is depressed beyond this is to be considered as flat-foot and rejected.

According to the severity of the symptoms, authors have divided the progress of a flat-foot into various degrees or stages:

1. Beginning cases; "Knickfuss"; pes valgus in its strict sense.
2. Inflammatory non-fixed cases.
3. Fixed or ankylosed flat-foot.

Another classification is:

1. Cases in which the arch only sinks on bearing weight.
2. Cases in which the arch is constantly sunken.
3. Cases in which the arch has become convex.
4. Fixation in the position of No. 3.

*Diagnosis.*—Where any of the typical objective symptoms are present or where the typical pains are complained of, the diagnosis presents no difficulties. Only those cases in which there are atypical

pains and few objective symptoms, or where these are out of all proportion to each other, or where the flat-foot is combined with or masked by other processes, can there be any difficulty in the diagnosis. It is always best to take a careful imprint of the bared foot, carrying the entire weight of the body, upon paper prepared in various ways, so as not to overlook that class of cases where the arch has become slightly weakened. (The simple imprint being all that the overwhelming majority of physicians will use, I will not mention the various methods and devices used for measuring, photographing, observing, or recording the angle, the amount of deformity, and the strength of the muscles.)

*Differential Diagnosis.*—Static flat-foot must be differentiated from all other forms of flat-foot in which the static conditions do not play the most important rôle; also from all diseases and conditions which can cause pain in the lower extremities and feet, the disappearance of the arch, and the deformity or change in gait.

Of first importance is rheumatism. In the sense as used by the laity and the majority of physicians (at least to their patients) the word rheumatism describes but a symptom — pain, stiffness, and discomfort in and about a joint. In this sense no differentiation can be made. If any objective signs of flat-foot be present the “rheumatism” may be ascribed to it.

Acute polyarticular rheumatic arthritis is an acute infectious disease, easily recognized in adults. In children the joint infections are infrequent. Acute rheumatism may, however, leave permanent deposits in one or more joints and the feet and ankles may be the seat of such residual deformity, as already spoken of under *Etiology*.

In arthritis deformans and chronic rheumatism similar changes may take place: the gait becomes shuffling, walking is painful and the tarsus swollen, but careful examination will reveal the general nature of the process. A soft, flexible support often brings relief to the foot symptoms.

Tumors, malignant or benign, syphilis of the astragalus or scaphoid, circumscribed swellings and œdema, abnormal bursæ and the like, may simulate a flat-foot in that the vault of the arch is filled up and that an imprint of the sole of the foot shows a marked flattening; pain and tenderness may come on early and the gait is altered. There will, however, be found a discrepancy between the

appearance of the foot, the apparent disappearance of the arch, and the real position of the bones of the foot. The relations of the internal malleolus, astragalus, and scaphoid will not be those typical of flat-foot. An X-ray photograph will often be of great aid in diagnosing bony growths and tumors.

Sprains and traumata may occur even relatively more frequently with flat-foot than under normal circumstances. The most important differentiation to be made is that between a sprained ankle of a flat-foot and a Pott's fracture. The crepitus, preternatural mobility, inability to walk, etc., of a fracture may be lacking here as in other atypical fractures. But here, as elsewhere, the four cardinal symptoms of fracture can always be elicited if looked for, viz.:

1. Pain on pressure definitely localized over the seat of the fracture.
2. Swelling of the character usually seen in fractures.
3. Bloody discoloration over this area.
4. Appearance of the parts in the X-ray photographs taken from two planes.

Angio-neurotic changes, as in the prodromal stage of spontaneous gangrene, Raynaud's disease, etc., must be carefully looked for in all cases where sweating and cyanosis are marked symptoms. The differentiation is often exceedingly difficult, and it will be found that most cases of Raynaud's disease, etc., have been treated for flat-foot at some time in the progress of the disease.

*Prevention.*—It is most important, yet exceedingly difficult, to prevent the occurrence of flat-foot; few cases ever come for medical advice until the condition has become painful. Wherever possible the physician should insist that growing children and young people, especially girls between the ages of 12 and 18, and those whose occupation demands excessive standing or walking, should be properly shod. Arches or supporting shoes should *never* be used, but, instead, a shoe with a broad toe, a straight inner edge (orthopædic last), and a low broad heel. It should fit snugly around the heel and instep, but be very loose and free at the toes. Almost all good infant's and children's shoes are now made over such lasts (Educator, Startrite, Walkrite, etc.). For men there are the various Inspector's, Orthopædic, Policeman's or combination lasts which fulfil the above requirements. For women fewer shapes are made; but in every



large city there will at least be one shop where such shoes as the Educator, Groundgripper, Walkrite, Health and Comfort, or nurse's shoes can be purchased. Care must be taken that the shoe salesman does not sell a long, narrow shoe when a shorter and wider size is called for (Fig. 36).

With increased demands for style and luxurious fashion, the physician will find it more and more difficult to persuade his patients to such shoes. The lady in Fig. 21 refused to wear the low-heeled shoes shown in Fig. 22.

Barefoot exercises, walking exercises, learning the proper poise of the body, exercise, and massage to the supinator muscles will be found useful to keep up the muscle balance of the foot.

Where there is a tendency towards pronation or eversion of the feet a Thomas heel or an additional half of a sole to the inner side of the shoe will be useful.

*Treatment.*—The treatment is necessarily divided into, first, the relief of the symptoms, and, second, the cure of the flat-foot. The first is easy; the second, difficult or impossible.

The symptoms can be promptly relieved by strapping the foot and ankle with adhesive plaster, so as to take the weight off the scaphoid and astragalus. The adhesive strap, two inches wide, should be carried from the outer side of the dorsum of the foot, around the sole and upward over the scaphoid, then backward and across the dorsum, and in a spiral manner around the ankle above the external malleolus and behind the calf, and then across the shins just below the knee. The foot should be actively supinated while the adhesive is being applied. The relief gained by thus supporting the arch is but temporary, and should never be used for more than a few days or weeks until the proper braces or shoes can be secured.

For the more permanent relief of symptoms in mild cases, or those whose feet can bear very little correction over an "active" brace, a balanced shoe with some form of arch support and stiff shank built in, as advised by Dr. Cook, of Hartford, Conn., will be found efficacious. In heavy patients the large projecting heel (Cook) or a wood shank filling up the space between sole and heel can be used (Figs. 37, 38, 39, and 40). The disadvantages of shoes as the basis of relief are:

FIG. 36.

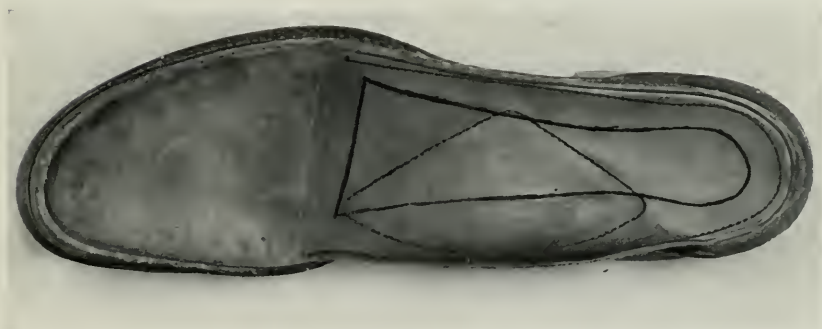


FIG. 37.



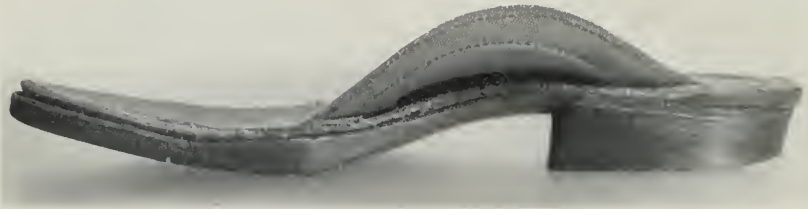
Fig. 36.—Broad weak foot. Note excessive length of narrow high-heeled shoe.  
Fig. 37.—Heel extended forward and to the inner side, the artificial but equally efficient equivalent of the turned-in great toe of the bare-footed savage (Through the courtesy of Dr. A. G. Cook, Hartford, Conn.)

FIG. 39.



Orange-peel instep support mounted on shoe bottom, showing position of steel shank. (Through the courtesy of Dr. A. G. Cook, Hartford, Conn.)

FIG. 40.



Orange-peel instep support, mounted on shoe bottom, showing heel. Lateral view. (Through the courtesy of Dr. A. G. Cook, Hartford, Conn.)

FIG. 41.

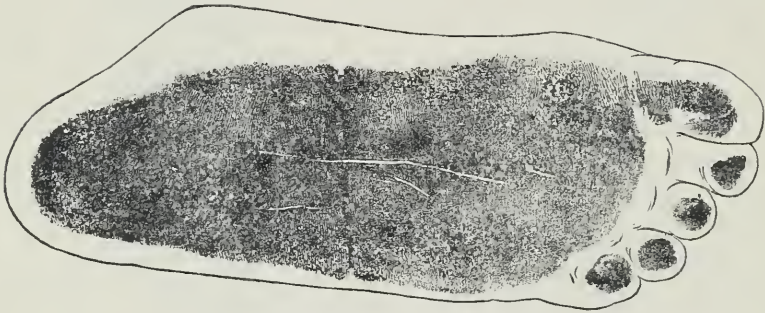


Fig. 41.—Blue imprint of severe case of flat-foot.

FIG. 42.



FIG. 43.

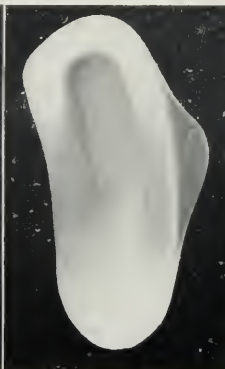


FIG. 44.



Fig. 42. Author's German silver flat-foot arch support. Side view.  
Fig. 43. Author's German silver flat-foot arch support. Bottom view.  
Fig. 44. Author's German silver flat-foot arch support. Front view.

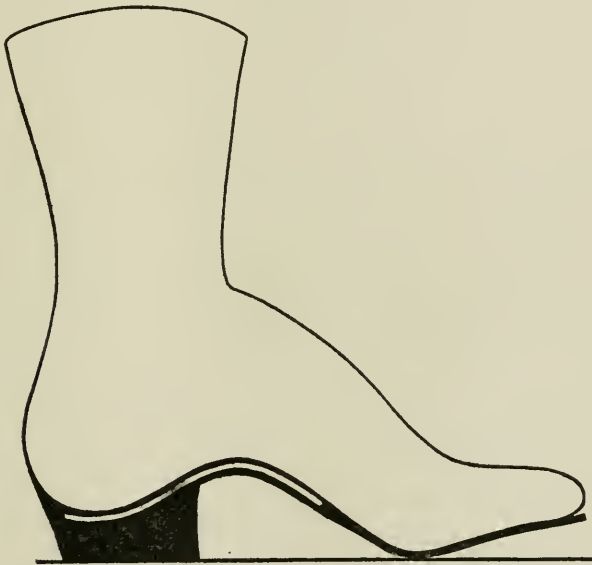
1. The cost of shoes is usually prohibitive to all those who earn less than \$30 per week.

2. It usually places the patient, his friends, and acquaintances in the hands of shoemakers for treatment.

3. Where once made, the shape and amount of support given by the brace cannot be changed. For each step in the treatment the patient needs a new pair of shoes.

4. When once thoroughly wet the leather becomes pliable and the shoes lose much of their supporting qualities.

FIG. 38.



The balanced heel, low. The stout shank iron, supported by the interior edge of the heel, supports the arch. (Through the courtesy of Dr. A. G. Cook, Hartford, Conn.)

Braces or arch supports are within the reach of all, will relieve symptoms, can be readily reshaped to meet the changing foot conditions, and can be worn in any appropriate shoe. Severe, tender, or ankylosed feet can best be relieved by beginning with some form of flexible, non-metallic arch support, which can readily be adapted to the needs of the patient from the many now on the market. Metallic arch supports are best made by a competent brace-maker after a pattern of the foot furnished by the physician. The best pattern is a plaster cast of the supinated foot, made after the manner described

by Whitman. Plaster cream just ready to set is poured into a paper-box cover and placed on a chair. The patient crosses one knee over the other and places the external malleolus of the crossed leg in the centre of the plaster, the foot being actively supinated. As soon as the plaster is hard it is wet with soapsuds and the rest of the foot covered with fresh plaster cream. When the latter is hard the upper half of the mold can readily be lifted off. It will be found best for the general practitioner to send this mold to the brace-maker, who can cast it, reconstruct the arch, and build the arch support accordingly.

Only the specialist or the more expert can shape the cast and reconstruct the arch successfully. If one has special confidence in his brace-maker it will be found just as efficient to take an imprint of the sole of the foot upon paper. White paper is moistened with a 10 per cent. solution of potassium ferrocyanide, and the sole of the foot is dampened with Tr. ferric chloride; the patient now steps firmly upon the paper, which yields a blue imprint of the bearing surface of the foot. Before the foot is lifted from the paper a projection is made by tracing the outlines of the foot on the paper by means of a pencil held perpendicularly. From such an imprint (Fig. 41) a competent instrument-maker can fashion an equally good arch support.

The type and form of metallic arch support to be used depend upon the training of the physician, the practice of the brace-maker, and needs of the case. There is no one type superior to all others; a leather covering has no practical value. My own practice has been to use a brace peened out on the anvil from cold-rolled German silver (40 per cent. nickel), which is elastic as steel, rust-proof, and light in weight. It is shaped without side hooks, to fit the entire sole of the foot up to the ball of the toes, and has a moderate but well-arched flange under the instep. There is also a transverse elevation to lift the metatarsal heads and raise the transverse arch and to allow foot to grasp plate so as not to slide off. Such supports must be reshaped from time to time to meet the varying conditions of untoward pressure, lack of support, etc. (Figs. 42, 43 and 44).

Where there is an unusual amount of pronation due to muscle insufficiency a corrective single-bar brace applied to the inside of the shoe will overcome it.

For the *cure* of mild cases of flat-foot, strengthening muscle exer-



cises with massage and a flexible shank shoe will suffice, if only the treatment be persisted in long enough. The patient must be taught to toe in, and in standing to put his weight upon the outer border of the foot. For the latter purpose the Thomas wedge-shaped heel finds its greatest use. As before mentioned, the exercises and proper posture must be persisted in at all times for at least two years. It is just in this respect that failure takes place, as few people are so strongwilled as to persist in the exercises in the hope of cure, when their neighboring shoe store is holding out a hope for relief for pain and discomfort (for a short time) for the small sum necessary to buy a pair of "Somebody's" ready-made insoles.

In growing children a metallic arch support as before described, made in an "active" pattern, *i.e.*, with the arch high enough to supinate the foot, combined with active exercises, will in time restore the position of the bones of the tarsus.

*Gymnastic Exercises.*—The following gymnastic exercises will be found serviceable:

1. The patient, with feet directed forward and slightly separated, rises and sinks on his heels, using the toes as much as possible.

2. The patient stands with the tips of the toes pointing as far inward as possible, with heels turned out, raises and lowers his heels, retaining the outward direction of the heels.

3. The same exercise is repeated, bending the knees as the heels are lowered.

4. The patient sits with outstretched knees and rotates the toes in a circle inward, downward, outward, and upward.

5. The patient walks on the outer borders of his feet as if he had club-foot.

Exercises should aim to attain a flexible foot and elastic gait. Hovorka makes his patients walk along a board six inches wide, planed to incline like a barn roof, with one foot on each inclined surface. Patients whose occupations make it impossible to avoid long standing should relieve the position from time to time by rising on the toes; they must realize that their own interest and will power must cooperate.

*Operative Treatment.*—Rigid flat-foot, although often amenable to a well-planned campaign with low arch supports, gradually raised, can most effectively be treated by forcible over-correction of the out-

ward rotation at the mediotarsal joint and the subsequent fixation of the foot in a plaster cast in the club-foot position. The adhesions between the tarsal bones must be well broken up before the foot can be over-corrected. The patient is to be encouraged to walk upon the over-corrected foot, and after from three to six weeks the casts can be removed and arch supports applied. Exercises must be used to preserve the flexibility of the joints.

Nicoladoni has advised an excellent bloodless operation for the relief of intractable flat-foot, which would otherwise be incurable. He cuts through the tendo Achillis just above the heel and reflects it upward, allowing the contracture of the plantar muscles and fascia to restore the arching of the foot, in the same manner that a "*pes cavus et contractus*" takes place in a case of paralysis of the calf muscles. He and his pupils cite many cases of immediate relief of bed-ridden patients and the restoration of their ability to walk many miles over rough roads a few weeks after this simple operation. The ultimate power of the patient to raise himself upon his toes does not seem to be seriously interfered with. Many other operations have been devised for the cure of flat-foot, but the writer cannot recommend them.

## DEATH FOLLOWING TWO CASES OF OPERATIVE FIXATION OF FRACTURE OF THE SHAFT OF THE FEMUR

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THE conversion of a closed fracture into an open one, for purposes of treatment, should be restricted to those lesions in which subcutaneous reduction of the fragments is otherwise very difficult or the maintenance of coaptation, previously obtained under anæsthesia, unsatisfactory. An unknown proportion of fractures should be subjected thus to incision, in order that proper replacement of the fragments may be obtained and direct fixation accomplished. This method has been advocated by me in various papers on fractures published since 1885.<sup>1</sup> It, therefore, will be understood that I am not opposed to the operative treatment of closed fractures in selected cases. There is, however, at the present time such an unwise advocacy of the open treatment of fractures that records of fatalities occurring from this method of treatment should be published. I therefore report two deaths following operative fixation of fracture of the femur with the hope that other surgeons may be influenced to do likewise.

The extensive urging of the operative management of these injuries, as an almost routine method, will lead to many fatalities in the hands of inexperienced operators. Many fractures of the bones of the upper and lower extremities are curable by non-operative means, provided that the surgeon has anatomical and mechanical knowledge. Such knowledge, combined with diligent attention to the treatment subsequent to the reduction of the fracture, will secure

<sup>1</sup> *Trans. Am. Surg. Assn.*, 1885, pp. 6 and 105. *Trans. Am. Surg. Assn.*, 1892, p. 58. *Annals of Surgery*, April 1895, p. 457. Paper read at the Brit. Med. Assn., 1898; published in *Phila. Medical Journal*, September 24, 1898. "Modern Treatment of Fractures;" D. Appleton & Co., N. Y., 1899. "Manual of Modern Surgery," Lea Brothers & Co., Philadelphia, 1899, pp. 364 and 369.

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a satisfactory cosmetic and functional result in most closed fractures.

The great activity in the operative treatment of closed fractures, which has developed within recent years, is due in part to the study of the broken bones by the means of the X-ray. This method of examination has vastly increased our pathological knowledge of bone injuries. It has, however, created a tendency to make the attending physician or surgeon neglect other clinical methods, which were formerly used to obtain a proper knowledge of the condition of the injured bones.

Other factors encouraging more frequent operative attack have been a general recognition of the safety of aseptic wounds, and the desire to avoid frequent visits and to lessen the time during which the activity of the patient should be restrained. Surgeons have become annoyed at the necessity for the frequent adjustment of splints and apparatus, which is demanded in the treatment of fractures of the extremities not subjected to operative management. An undue reliance on the supposed truthfulness of even carelessly taken X-ray plates and an indolent neglect of fractures, treated by external appliances, have given rise to a belief that many fractures are curable only by exposure of the bones, and fixing the fragments with screws, wires, or metal plates. These operations are now being attempted by men who have not had sufficient surgical experience to be entitled to confidence when they attack deep-seated injuries of the skeleton. Good results with less risk to life may often be obtained by less dangerous methods.

The determination of whether a fracture should be subjected to operation by studying X-ray plates alone, leads at times to unnecessary operations. X-ray plates of fractures are of real value only when carefully and properly made by an expert radiologist, who is also somewhat acquainted with the surgical problems demanding solution. The plates may easily deceive the surgeon by giving an erroneous impression of the osseous relations. They should be used only as collateral evidence in connection with other diagnostic and therapeutic methods at the disposal of the surgeon. Stereoscopic plates, or plates taken in two planes at right angles to each other, may be required to furnish the information which the surgeon needs. They should be made before the fracture apparatus has been applied

and also while it is being worn, if they are to be used to disprove the probable success of the non-operative treatment.

It seems often to be forgotten by surgeons that no accurate determination of the degree of shortening in fractures of the extremities is possible, unless there exist previous accurate measurements of the two extremities before the fracture occurred. Asymmetry in the length of long bones is a very common condition. I proved this in 1878<sup>2</sup> by measuring the bare bones of skeletons having normal femurs. This inequality in length has been repeatedly shown since that time by other investigators. To ascribe to imperfect treatment, non-operative or operative, a moderate degree of shortening in a leg or arm after fracture is a mistake, unless the exact equality in length previous to injury has been established. This, naturally, can scarcely ever be done in practical surgical work.

One sees reports of fracture of the femur treated by operation, because of the asserted inefficiency of its prior non-operative treatment. In some of these cases the prior treatment apparently has been conducted without giving the patient the benefit of strong continuous traction and countertraction by Buck's method. The muscles of the thigh are so powerful and usually cause such overlapping of fragments, when the femoral shaft is broken, that I doubt whether a surgeon is justified in adopting the open method unless he has first employed, for a couple of days at least, continuous traction and countertraction with from 15 to 40 pounds by the Buck method.

When a fracture of a long bone can not be satisfactorily reduced or when, if it can be reduced, maintenance of the coaptation is impracticable, it is proper for a trained aseptic surgeon to operate. It is essential, however, that he or a similar expert have charge of the after-treatment. The operation should be done only when asepsis can be obtained, and be maintained after operation; and it is important that the surgeon and the attendants have expert knowledge and be willing to give the time to avert any secondary septic symptoms.

Fractures of the shaft of the femur just below the small trochanter, very oblique fractures of the shaft, and fractures at the lower

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<sup>2</sup> *Philadelphia Medical Times*, August 3, 1878.



end of this bone demand operative fixation with more frequency probably than other fractures of this or other long bones. (Fig. 1.)

The non-operative method is usually sufficient to gain a good result in other fractures of the femur and in fracture of other bones of the extremities, provided that the surgical attendant knows the anatomy of, and the mechanical problems presented by, the injury,

FIG. 1.



Diagram of X-ray plate of fracture of the femur, which is difficult to keep in position by non-operative means.

FIG. 2.



Diagram of X-ray plate of fracture in the case of J. D.

FIG. 3.



Diagram of X-ray plate of patient who refused to have fracture through neck of femur nailed.

gives proper attention to the case, and supplements the want of knowledge of the other attendants upon the patient by his own surgical acumen and vigilance.

The operative treatment of fracture of the femur is more difficult than the operative treatment of fractures of the bones of the leg or of the upper extremity; but in selected cases it may be required. In some other cases it may be preferable to non-operative treatment,

if the patient can be given the benefit of the proper environment and skill.

Direct fixation by operation of fractures of the large and deeply situated bone of the thigh should be undertaken with great caution in the average American hospital, because of the imperfect organization of many of these institutions. The short terms of unsalaried individual surgeons, the imperfect training and service of the usual resident physicians and nurses, and the dangers of prolonging anaesthesia in unskilled hands make this method too risky, when compared with the non-operative method. These faults in hospital organization militate somewhat against satisfactory results in non-operative cases also; but the danger to life is much less in the latter than in the former instance.

I report at this time two cases of death occurring after open treatment and fixation by plates of fracture of the shaft of the femur below the small trochanter. I do this to show that such operative treatment is accompanied by risks that ought to be well considered by surgeons before deciding upon its adoption. In both of these cases I concluded to operate, because I believed it was likely that I could do better for the patient by rejecting non-operative measures. I thought I was giving the patient the best chance for a good leg and not especially imperilling his life. The result showed that I was mistaken.

I am quite sure that if all cases of death following the operative treatment of this important injury were recorded publicly, the medical profession would be convinced that there is a considerable risk accepted by a patient who permits his surgical attendant to open a closed fracture of the femur and treat it by direct fixation.

#### TWO DEATHS FROM SUBTROCHANTERIC FRACTURE OF THE FEMUR TREATED BY DIRECT FIXATION

H. H., aged 50 years, who had a history of alcoholic addiction, though recently he had abstained from the use of alcohol to a great extent, on April 27, 1911, tripped over the carpet and fractured the left femur below the small trochanter. He was admitted to the Polyclinic Hospital on the same day and the site and character of the fracture were confirmed by an X-ray plate. The upper fragment was

tilted upwards by the action of the psoas and iliacus muscles. His temperature was about normal and the pulse about 80; respirations about 20. After treating the fracture for two days and finding it difficult to obtain apposition by Buck's extension and the inclined plane, I determined to open the soft tissues and to apply a metal plate to the fragments for fixation. Accordingly, on April 29th, I made an eight-inch incision on the antero-lateral aspect of the left thigh and exposed the broken bone. By means of extension, applied with compound pulleys attached to the stirrup of the Buck's apparatus, the two fragments were brought into apposition and two rectangular metal plates adjusted, so as to fix the fragments in good position. One of the plates was put on the anterior surface of the bone, the other alongside of it, but more on the lateral aspect of the femur. The latter was twisted so as to give a hold around the shaft of the bone in a manner to prevent lateral as well as antero-posterior displacement. The wound was washed out with normal salt solution, the muscles approximated with interrupted catgut sutures, a gauze drain carried down to the site of the operation and the skin closed throughout the rest of the wound with silk-worm gut. A gypsum encasement splint was applied and the limb supported on an inclined plane with Buck's extension added.

Within a few days the gauze was removed and the same line of treatment continued. Suppuration took place and the wound was then treated through a window in the gypsum splint; being irrigated with solutions of formaldehyde, and later with other antiseptics, for the purpose of averting septic processes in the wound. The temperature at the time the drain was taken out, which was about three days after operation, had risen to  $101^{\circ}$ . It fell to the normal and continued at about that point. Later the temperature rose; and an abscess was found in the wound. It was opened and the temperature curve again fell to about the normal line. In June, I transferred the patient to my colleague, Dr. Jopson, and asked him to remove the plates, as I was about to go out of town. At that time there was a sinus at the point of drainage from which pus was escaping. He opened the wound, found the fragments in good position, and took out the plates, but the patient died on July 5, 1911, without having any special rise in temperature. No autopsy was obtained.

FIG. 4.



Vicious union right humerus. Treated by osteotomy and nailing with fracture-nail in March, 1901. Wire attached to head of nail and brought out of wound to facilitate search for nail when it was to be removed. Nail removed at 5 weeks. Dr. Roberts's case.

FIG. 5.

Seat  
of  
fracture



Dr. Roberts's patient, S. D., operated upon February 26, 1908. Great anterior deformity, due to malunion of fracture below small trochanter, caused by flexion and outward rotation from action of ilio-psoas muscle. Resection of the ends of fragments and fixation with Roberts fracture-nails. Cured with good limb. Illustration shows the manner in which X-ray plates are deceptive unless taken in two planes or stereoscopically. Here there is scarcely any appearance of fracture or of displacement shown.



I believe Dr. Jopson considered the death to be due to exhaustion secondary to chronic alcoholism. During the entire time there were evidences of what I first looked upon as alcoholic delirium, due to the patient's previous life. The man was restless at night, wandered in his conversation, but showed, if I recollect clearly, no albumin in his urine. Unfortunately the notes of the urinary examination were not preserved. Thinking that the solutions of formaldehyde used to irrigate the sinus and to dress the wound might be the cause of the mental aberration, I substituted other antiseptic washes. The man's condition did not seem to be modified by the change.

J. D., aged 60 years, whose general health was good, although he had evidence of some hardening of the arteries, fell on the street, January 31, 1912. He struck on his right hip and fractured his right thigh bone a short distance below the smaller trochanter, with the upper fragment split longitudinally upwards. (Fig. 2.)

The skiagraph shows an inverted V-shaped line of fracture running up into the neck. The patient's appetite was good, his bowels were regular, his thoracic organs seemed healthy on examination, he was alert in mind and there was no albumin in his urine. He denied alcoholic addiction.

After endeavoring to keep the fragments in position by elevation on an inclined plane and Buck's traction method, I found that good apposition was not obtained, although I used 35 pounds weight upon the stirrup of the Buck's apparatus. After giving the patient's condition careful consideration, and concluding that he was in even better health than I at first thought, I determined to make an incision and to try to reconstruct the bone by applying a Lane's plate to the broken femur.

Through a long incision it was comparatively easy to apply a Lowman clamp, so as to hold the Lane plate and two fragments together. The shaft and the great trochanteric fragment were then fixed with the plate. Six screws were used. They were long enough to perforate the shell of the bone and pass through the marrow. They probably reached the opposite side of the bony cylinder. The other fragment of the upper end of the bone was so deeply buried in the muscles at the inside of the thigh that no attempt was made to fix

it with screws or plate. It could probably have been done with a drill-fracture nail. Because of the great damage done to the soft parts by the original injury, I thought it wise to leave a gauze drain in the wound between two of the sutures. A wet dressing of liquor formaldehydi (1-500) was, I think, applied. Buck's traction apparatus was used with a few pounds weight, with the limb extended on the bed without the inclined plane, which had previously been employed. A gypsum encasement was not employed because of my belief that there probably would be suppuration of the wound as a result of the extensive extravasation of blood.

This operation was done at the Polyclinic Hospital eight days after the injury. The patient's temperature after operation remained nearly normal for several days, varying from about  $97^{\circ}$  to  $98\frac{1}{2}^{\circ}$ . The pulse ran from about 80 to 100; the respirations from about 20 to 24. On the third or fourth day the wound was dressed and the gauze drain removed.

Nine days after operation I found that there was pus in the wound, though the temperature had not gone very high, being about  $100^{\circ}$  to  $101^{\circ}$ . On removing the stitches and separating the wound I evacuated about 4 fluidounces of pus. This occurrence induced me to introduce a rubber tube, which I carried from the lower part of the wound under the central stitches and brought out at the top of the wound. Irrigation was done daily with water and liquor formaldehydi (1-500), and the same strength of the antiseptic was used for an outside dressing. The strength of this solution was at times made weaker. Frequent dressing and covering the wet gauze with waxed paper was necessary, because in using the urinal the man frequently saturated the bandages with urine.

About this time some impairment of resonance of the right side of the chest was noticed by me. Dr. David Riesman, my medical colleague, examined the man a few days later and confirmed the diagnosis of impairment of resonance of the right side, which he thought to be pulmonary and not pleural. He also thought there were some implications of the pleura on the left side. This condition was treated. Some time later the patient showed hebetude and had a very coated, dry, furred tongue. He presented an appearance very much like that of typhoid fever, but without rise of temperature. His condition suggested to Dr. Riesman a catarrhal pneumonia

with pleurisy and perhaps a uræmic state. The man then began to pass urine in the bed unconsciously and lapsed into a dull mental condition very different from his original alertness of mind, and died February 29, 1912.

About the time that the wound showed so much infection he soiled the dressing on several occasions by spilling the contents of the urinal over his thigh. Urinary examinations were made quite frequently, and some time after the operation a slight amount of albumin and a few casts were discovered. The patient also became delirious at times and rapidly lost flesh. Later the percussion note over the right lung seemed to be almost normal, and the left lung on ordinary examination appeared to be free from disease. There was very little rise in temperature during all this time, though occasionally it reached 101° or thereabouts. His pulse remained at about 80 or 100. His respirations during the time he had the more active lung symptoms went up to about 30 per minute, but they were generally about 20 to 24 per minute.

I had watched the wound carefully and found that there was no discharge of pus after the first few days, subsequent to the opening and draining of the wound, but the man's general condition was very unsatisfactory. I could detect no evidence of burrowing of pus and no active suppurative symptoms. I considered and discussed the propriety of opening the medullary cavity of the bone with a trephine during the latter part of his life, but was deterred from such operative treatment because the emaciated limb, the normal temperature, and the freedom from pain, swelling or other evidences of suppuration in or around the bone convinced me that the wound was doing well.

My surgical colleagues, Dr. Morris Booth Miller and Dr. John H. Jopson, saw him and aided me with suggestions, but the man died on February 29th, twenty-one days after operation. There was a slowly increasing failure of vital powers with hebetude, increasing to coma, suggesting uræmia or septic processes. The cause of the toxæmia, I could not explain to myself, although septic conditions in the wound seemed more probable than uræmia or alcoholism alone. There was no color evidence of the blue pus of pyocyaneus infection observed.

No autopsy was allowed by his friends, but Dr. Rinker, of the resident medical staff, who removed the plate from the wound, reported the following local conditions: Two large pockets of pus were found, one extending up to the groin near the inner surface of the thigh; the other downwards along the inner part of the thigh to about its middle. When the plate was removed the fragments separated and the marrow cavity of the upper fragment contained bloody purulent material. The soft tissues of the incision had united with the exception of the drainage opening, but the muscles and interstitial tissues of the inner aspect of the thigh were in a necrotic state. The contents of the two pus pockets did not look like pure pus; the fluid was more viscid than true pus. It was of a dark reddish color and contained the débris of necrotic tissue. The capacity of the upper pocket was about five fluidounces.

Study of these two cases shows, I think, that the latter died of septicæmia arising from purulent inflammation in the bone marrow and in the tissues around the bone; and that the former probably succumbed from the same cause, though chronic alcoholism may have been a factor in that death. The symptoms were very similar, except that the first man died about three weeks after operation, the second lived nine weeks. Both were laboring men beyond middle life and therefore perhaps injudicious selections for the so-called "bloody" treatment of femoral fracture.

I, however, decided to operate upon each of them instead of adopting non-operative methods, because I realized that a laboring man finds a deformity or an incapacity from fracture of the femur a greater handicap to his career than would a patient belonging to a higher social group. Both stood the shock of operation well. I watched both carefully; one until death, the other until shortly before death. I transferred the latter to the care of a well-known skilled surgeon. Nevertheless, death came to both, because I fastened the displaced fragments together by direct operation, thereby converting closed fractures into open ones. Perhaps vertical traction such as we use in fracture of the femur in children or the Smith or the Hodges anterior wire splint would have given a good apposition of fragments.

It may, however, be said that they might have died under non-operative treatment. Such instances are known to most surgeons.

FIG. 6.  
a



FIG. 7.  
b



Staples applied to comminuted fracture of tibia. The legs of staples were too thick and did not go down in drill holes far enough to bring staples close to surface of bone. Dr. Robert's patient, P. G. Operation, April, 1905.

Result in P. G.'s case of comminuted fracture of tibia and fibula five months later.





While I had the second case under my care a woman of about 50 years was admitted to my ward with a badly comminuted fracture of the cervical and trochanteric region of the left femur. I was unable to keep the fragments in proper apposition by various external fracture dressings, which I tried, and then advised her to let me drive a nail or screw through the large trochanter for fixation. (Fig. 3.) She refused. Non-operative treatment was therefore continued. She became stuporous and delirious, removed her dressing, and had unconscious urination. As a result she developed bedsores and presented almost indential symptoms with those of the two men whose death I report. She is now slowly recovering.

During the same period of time one of my patients, who had received a bad spiral fracture of the left femoral shaft, while suffering from a gonorrhœal discharge and cystitis, unexpectedly died, within a day or two, with pulmonary symptoms, although he had been convalescing satisfactorily for a couple of weeks. The man's improvement in the initial symptoms of the fracture, the decrease in the discoloration of his thigh, which had been caused by the sub-muscular and subcutaneous extravasation of blood, my frequent examinations and his general aspect of doing well gave no warning. Unfortunately no autopsy was obtained, but clinically the death seemed due to pneumonia from influenza infection. Fat embolism did not seem likely.

It is also proper for me to recollect that operative attack on the femur may be followed by fatal surgical shock. A dozen or more years ago I had a young girl of 16 or 18 years of age die a few hours after excision for ununited fracture of the shaft of the right femur. She was in perfect general health and had previously been operated upon in another city for non-union of the broken bone. That operation had failed to induce union. She was very much frightened when she came to the operating room and I myself started the anæsthesia. I resected the ends of the fragments so as to make a mortise and used screws or wire to hold the bone ends in apposition. The procedure was rather long and difficult, but she did not lose too great an amount of blood and was not badly etherized. Still, while on the stretcher on the way to the ward she showed suddenly signs of collapse and died in a few hours despite my best efforts to cause reaction. I think that she was carried upstairs head first, which

may have caused injurious cerebral anæmia. As is so unfortunately and so commonly the case in our American surgical work, no autopsy was obtained to determine the psychic or anatomical cause of death.

As I have already said, a purpose in reporting these deaths, after the now fashionable operative treatment of fractures by direct fixation, is to neutralize, if possible, the effect of the unwise enthusiasm of some surgical writers.

I admit that in my cases I may have been mistaken in employing drainage for a few days; the perforation of the marrow cavity of the bone may have invited unnecessary medullary sepsis; the dressings of formaldehyde, after the wounds became infected, may have given rise to toxic symptoms through absorption. Still, the fact remains that death took place in two patients under the care of a surgeon who thought, perhaps presumptuously, that he had sufficient experience to undertake the work and to decide on its advisability.

For years I have advocated the open treatment of broken bones in selected cases of closed fracture, and have occasionally resorted to the method. This recent experience has nevertheless proved to me that our profession must be guarded in its advice relative to the general adoption of operative methods in the management of fractures. Some of these injuries demand operative treatment, some will permit its employ in competent hands and under proper surroundings; but others will be followed by unnecessary fatalities. In the last group, some of the patients will have a better chance for life if less strenuous methods are used by the surgical attendant.

The paper is a plea, if I may use that term, for a conservative progressiveness in the surgical art. Enthusiastic adoption of the newest suggestion may lead to disaster, if not restrained by conscientious consideration, careful observation and logical thinking. I have no words but condemnation or pity for the obstinate, unseeing, and unhearing "holdback" in science; but unthinking, rash, and dogmatic statements without the straight thinking, recently so well advised by a well-known college professor, cause in many minds a psychic emesis. They may even lead the hearers to become too conservative, and reject the seeds of truth; and thus do harm. They certainly in surgery will, as they have heretofore, fill untimely graves.

# THE LORENZ OPERATION FOR CONGENITAL DISLOCATION OF THE HIP

BY JAMES K. YOUNG, M.D.

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THE technic of Lorenz's operation for congenital dislocation of the hip, its rationale, and the results obtained by this manual method of reposition are already well-known facts in the annals of surgery. Suffice it to remark that the number of cures effected through this procedure is constantly increasing, so that the grand total of perfect results obtained is far in advance of antecedent methods that depended upon the employment of various surgical measures for the relief and cure of this affection.

Like all new measures, the Lorenz operation was at first received with some degree of conservatism that gradually gave way to expressions of scepticism, whilst at the very outset not a few surgeons denounced this new procedure with emphatic disapproval. But as the beauties of the new operation were unfolding themselves, and as the results disclosed many perfect cures, some of the medical profession began to realize the Lorenz method to be a great addition to the domain of orthopædic surgery and far superior to the open methods that statistically gave varying degrees of success.

For fifteen years Lorenz had been making an exhaustive study of the subject. In fact, Bradford of Boston, Hoffa of Berlin, and Lorenz of Vienna formed a surgical triumvirate, and they announced that they had perfected an open operation for the treatment of congenital dislocation of the hip—when just at the psychological moment word was flashed over the civilized world that Lorenz of Vienna had devised a bloodless method for the cure of this class of affections that would afford more successful results than those then in vogue.

Much of the disapproval expressed and implied was not so much caused by the new method of reposition as it was by the lack of

knowledge of many of those who interested themselves in the then recent discovery. All kinds of opinions ran rampant. Some practitioners attempted the reduction with little or no experience with deformities. One surgeon consulted me for the technic, admitting that he had never performed the operation, nor had he seen it performed. A gynaecologist and general surgeon wrote to me for details, as he had previously accepted an invitation to read a paper on the subject, informing me at the same time that he had never seen the operation, nor had he read anything about it. Thus, confusion arose in the conflicting reports that were heralded broadcast. Every one seemed to offer an opinion at once before allowing sufficient time to judge of the results attained. The tyro in the art was largely responsible for the dissension and discussion that almost at once arose when Lorenz first demonstrated his masterpiece in surgery before the public clinics.

*Diagnosis.*—An accurate diagnosis is, of course, imperative. The head of bone must be present and not deformed; the upper rim of the acetabulum must be present, and the age of the child is, of necessity, a governing factor. To perform this operation successfully, one must be thoroughly familiar with the anatomy and the mechanics of the hip-joint and should have attained some degree of skill in the treatment of orthopaedic cases. The one important fact that must never be lost sight of is the orderly sequence of the movements as enunciated and practised by Lorenz, which must be rigidly adhered to.

It would be a superfluity to rehearse the commonly-known facts relating to congenital dislocation of the hip, but it seems pertinent at least to review very briefly the mechanism concerned in this variety of dislocation as it is revealed in autopsies upon the new-born. In this class of cases the acetabulum is rudimentary, elongated and more narrow than normal, covered with hyaline cartilage, and perhaps filled with fat and fibrous tissue. When the upper border of the cartilaginous rim is lacking, the cavity merges with the surface of the ilium. The head of the femur, although atrophied, is slightly larger than the concavity of the acetabulum, whilst the neck of the bone appears shortened and conical and forms a less obtuse angle at its junction with the shaft of the femur. The round ligament is filiform and attenuated, and this same attenuation is well marked at the



capsule of the joint, where the enlarged cavity is filled with an excess of synovial fluid. The abductor muscles in relation to the joint undergo atrophy and the adductors retraction, whilst the pelvis suffers a lateral compression with a contraction of the superior and inferior straits.

When the dislocation is unilateral the pelvis is usually somewhat atrophied on the affected side. In the bilateral variety the pelvic changes are produced by the traction of the muscles, especially the psoas, and of the ligaments in a transverse direction, lessening the inclination of the pelvis and producing lumbar lordosis, due to the changed positions of the heads of the femora and contraction of the adductors.

The diagnosis of congenital dislocation of the hip offers no difficulty; nevertheless, in the very young (before walking) the affection is not infrequently overlooked. Briefly considered, the essential factors constituting the case include the patient's history, the prominence and elevation of the trochanter above Nélaton's line, the abnormal mobility of the joint, and the alterations in the width of the pelvis. A corroborative measure of the greatest importance is the diagnosis by radiophotography, which will clearly demonstrate the character of the disability and differentiate it from other affections.

*Differential Diagnosis.*—There are a number of diseases and affections that more or less closely simulate the symptomatology of congenital dislocation of the hip, the more common of which are:

1. Epiphysitis.
2. Coxitis.
3. Infantile paralysis.
4. Spontaneous dislocation.
5. Traumatic dislocation.
6. Coxa vara.
7. Fracture of the neck of the femur.
8. Pseudomuscular hypertrophy.

1. *Epiphysitis* (Fig. 1).—The resemblance of the deformity of unilateral epiphysitis to unilateral dislocation is extremely marked, and where there has been no abscess formation followed by a cicatrix the differential diagnosis must rest upon the skiagraphic findings. Before the child is two years of age the diagnosis is impossible, even

with the use of the skiagram, because the centre of ossification in the head of the femur does not form before this time. Subsequently the differentiation can be easily made out (Fig. 2). Where scars about the joint point to the existence of former disease and a clear history is available, the differentiation is readily established, or the diagnosis may be evidenced by finding the remains of a similar lesion in another part of the body.

2. *Coxitis* bears some resemblance to congenital dislocation of the hip, in that the patient suffers a persistent limp and, in the later stages, there is shortening, but in the latter affection the limp is congenital, there exists excessive mobility of the joint, and there may be noted complete absence of reflex muscular spasm or limitation of motion. Congenital luxation is more frequently bilateral than is coxitis.

3. Cases of *infantile paralysis* present the peculiarity of gait, the inequality of the limbs, and the laxity of the hip-joint, all of which symptoms may strongly simulate congenital dislocations; but the inroads of infantile paralysis manifest themselves by the cold and atrophied limb, the laxity of all its joints, and especially by the responses elicited by the passage of the electric current.

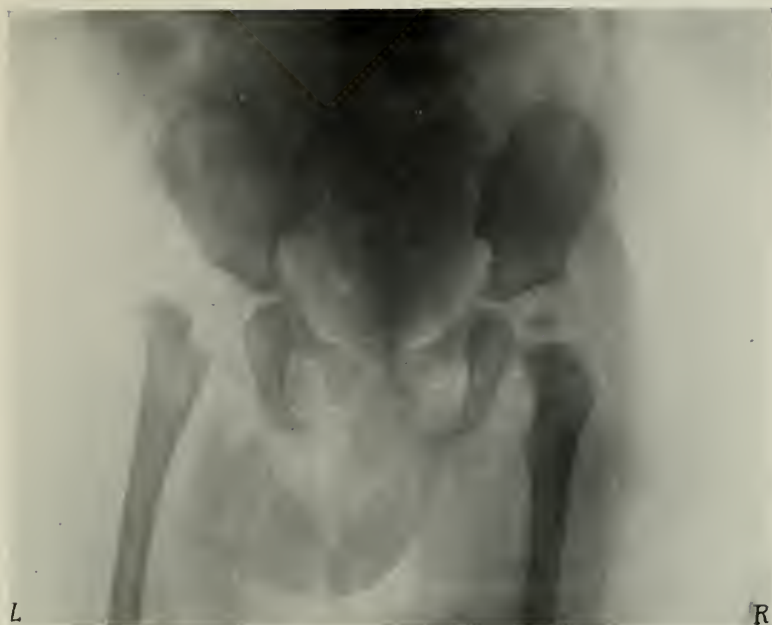
4. *Spontaneous dislocation* of the hip may be differentiated by its history. It occurs at times in association with acute synovitis; the occurrence of luxation predisposing to repeated luxations. This accident frequently follows in the wake of attacks from rheumatism, scarlatina, typhoid fever, etc., and it may be a sequel to infantile paralysis and occur late in hip-joint disease.

5. In *traumatic dislocation* there is offered the history of an obstetric injury and the fixation of the joint. The stability of the joint following reduction will confirm the diagnosis.

6. In adolescent *coxa vara* of rhachitic origin differentiation is to be arrived at by the following distinguishing characteristics: *Coxa vara* is usually acquired, whilst in congenital dislocation of the hip the abnormal mobility dates from birth, and upon palpating the head and neck of the femur by forced flexion and adduction of the thigh the trochanter will be found freely movable beneath the tense tissues of the gluteal region.

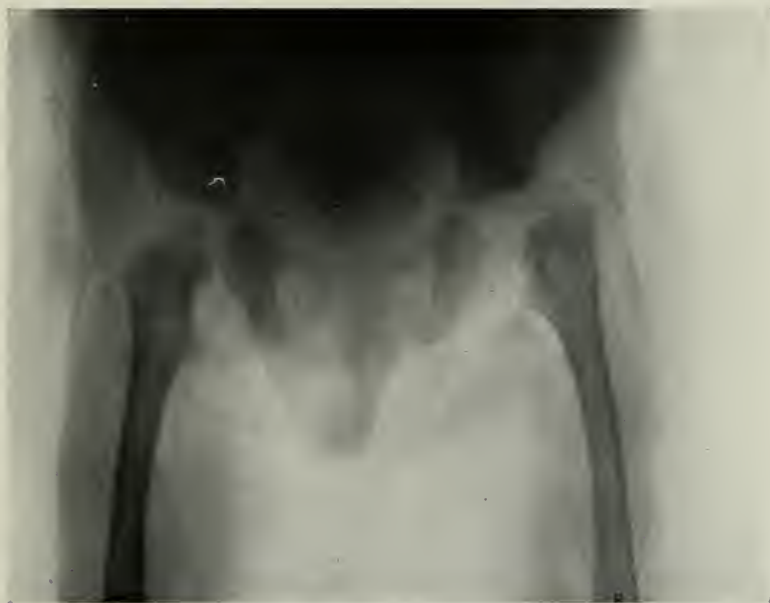
7. *Fracture of the neck of the femur* in children (traumatic coxa vara) is separated from congenital dislocation by the history of the

FIG. 1.



Unilateral, congenital dislocation; early stage. Note the absence of the epiphysis; compare with Figs. 3 and 4, taken from same patient.

FIG. 2.



Unilateral epiphysitis, showing destruction of epiphysis. (Posterior view.)

FIG. 4.



Unilateral dislocation following reduction.

FIG. 3.



Unilateral dislocation, showing presence of epiphysis. *Vide* Fig. 1.

injury, shortening of the limb, some restriction of motion, but a greater limitation of movement in the acts of flexion, abduction, and inward rotation. This fracture can be positively diagnosed only by a skiagram.

8. *Pseudomuscular Hypertrophy*.—In this affection the lordosis and the impairment of locomotion somewhat resemble congenital hip dislocation; but the latter condition does not exhibit the normal position of the head of the femur, the excessively-developed calf muscles, and the atrophy of the latissimus dorsi, teres major, and the lower portion of the pectoralis major muscles (Figs. 3 and 4).

TECHNIC OF REDUCTION.—To obtain successful results, it is absolutely essential that the different steps in the operation must follow in the orderly rotation enunciated and practised by Lorenz himself. These procedures are as follows:

1. *Hyperabduction* and tearing of the adductor muscles. With the pelvis fixed, the surgeon produces a forced abduction of the limb, and by manual means detaches the adductor muscles from the pelvis, unless they have been previously tenotomized.

2. *Hyperflexion*.—This second manœuvre is accomplished with the patient in the prone position, whilst the lower extremity is slowly but forcibly flexed until the foot touches the ear of the corresponding side, resembling the position attained in the operation of stretching the sciatic nerve. This is the movement in which the femur may be fractured unless great care be exercised.

3. *Hyperextension*.—This third step demands that the patient should repose on the opposite side when the surgeon practises forcible extension of the lower extremity while the knee is flexed.

4. *Traction*.—This, the fourth part of the operation, requires the patient to be placed in the prone position whilst manual traction is exerted upon the extremity. Or a skein of yarn is secured about the ankle, the surgeon thus effecting traction, as the assistant maintains the pelvis in a fixed position.

5. *Reduction*.—In this, the final step, a triangular wooden (Koenig's) block is placed beneath the trochanter, the patient is in the prone position, the thigh is strongly abducted and the hip reduced. With the reduction of the head of the femur, hyperabduction is practised, together with rotation, and the anterior part of the capsule is thus enlarged. To prove the occurrence of reduction it is only neces-



sary to observe that the knee-joint cannot be extended beyond a right angle.

In a brief article such as this it has only been the desire to emphasize some of the more salient measures essential to the successful performance of the Lorenz operation, and this affords an opportunity for a thought or two on the all-important subject of the after-treatment of this class of cases.

**THE AFTER-TREATMENT.**—The fixation bandages or cast which maintain the limbs in a hyperabducted position with the knees flexed are replaced at intervals during the first six months, but retain the same position. At the expiration of six months the limb is placed in a position midway between abduction and adduction, and midway between flexion and extension. In those cases where a block has been employed (that is, in unilateral cases with a high shoe, elevated four or five inches by a block) the height of the latter should be correspondingly reduced until it is only about two inches in thickness.

At the end of one year all the casts are discontinued and the patient will walk with the limb extended, but there still will remain eversion of the foot for nearly another year. During the waking hours a splint is used, but it is provident to employ a removable abduction cast at night in order to forestall any tendency to relapse.

It is at this period that carefully-selected gymnastic exercises and massage are very advisable as conducing to the best possible results. The pelvic trochanter muscles, especially the abductors, require certain set exercises, and, as is well known, varying gymnastic movements are demanded, depending upon whether the dislocation is unilateral or bilateral. The employment of massage is indicated after the removal of the second fixation bandage, and that treatment is most efficient that combines with its massage the movements of friction and kneading.

For a period of two years after reduction the patient walks about with the foot everted. The normal reposition of the foot in locomotion may occur incidentally, or it may be necessary to educate the child as to the manner of walking properly. When the reduction is permanently established, it is advisable to allow the foot to adjust itself normally, *i.e.*, approaching the frontal plane.

The Lorenz method of treatment occupies a period of two years' duration, about the same length of time consumed by the older trac-

tion method of Brown. But in the more modern method (the functional weight-bearing) the patient brings the limbs into play two or three weeks after the reduction, and thus the natural muscular tone and contractions are more rapidly restored.

The final results obtained by the Lorenz method are indeed illuminating. Some years since Lorenz reported 50 per cent. of functional cures by this operation, and I have no hesitancy in stating that at the present time the statement of the same estimate is in no way exaggerated. If anything, the number of cures shows a progressive increase. The bloodless operation for reposition of the hip is now cordially approved of by even the most radical surgeons; the new method has stood the test of time as it has stood the test of intelligent criticism. The procedure is dangerous and ill advised when attempted by those not conversant with the subject, or by operators whose experience in this field of endeavor is limited.

## BREAST MALIGNANCY: A PLEA FOR EARLY OPERATION AND EXTENSIVE DISSECTION

BY GEORGE S. FOSTER, M.D.

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DURING the past decade malignant growths of the female breast seem to have been on the increase. A definite statement to this effect could be made if clinical records alone be considered. However, it seems to me that we have no reason to believe that there is actually greater frequency of these cases. The truth is that the women of the present generation are becoming better informed along these lines, and therefore go to their family physician earlier for treatment than was the case even five years ago.

This brings results with which all surgeons should feel pleased. When we see the cases early we at once feel more sanguine as to the prognosis. We immediately decide that here is a patient whose life we can save by an early operation. If a patient steps into our office with her story of having recently noticed a bunch coming in her breast we at once, almost intuitively, are aware that we can deal with this person satisfactorily. It is the older cases, those who have kept the secret from the members of the immediate family and even the health adviser, with which we cannot be so successful.

This sort of a case brings to us some concern as to just how much we are able to do for the patient in a surgical way. We are given a history of more than six months' duration; in fact, it may be one or two years in length. Examination reveals an ulcerated area upon the periphery of the areola, beneath which we can palpate a hard nodular mass. The axillary, infraclavicular, and sternal glands may or may not be palpable. Should these little fighters of the breast war be present we know that a desperate battle is ahead of us. We,

as surgeons, at once realize the responsibility of our position and we give it deep thought.

We should, therefore, always be careful to inform our younger patients of the dangers of breast disease if it be neglected. No heroic act deserves greater credit than the early removal of a breast-tumor. The papers announce that a medal of honor has been awarded this or that person for some great heroic deed, the saving of one or more lives by physical effort. Carnegie medals are annually distributed, yet it remains for one of these to some time be awarded a member of the surgical world for the heroic act which is not infrequent in any surgeon's life. However, after all, we do not do our work with the idea of receiving medals of honor. A surgeon does his duty for the purpose of relieving suffering humanity, regardless of remuneration.

#### MAMMARY GLAND ANATOMY

The mammae are accessory glands of the generative system, which secrete milk. The gland, so important to the female, is hemispherical in shape, situated toward the lateral aspect of the pectoral region, corresponding to the interval between the third and sixth and seventh ribs. The weight and dimensions vary at different periods of life and with different individuals. Thus situated, the gland is the more exposed to traumatism and continued irritation than any other important part of the human anatomy, unless it be the eye.

Previous to puberty these organs are small and flat, but as the generative organs develop the breasts become more prominent and void. They complete a cycle, increasing more extensively during pregnancy following delivery, and so on until they undergo atrophy at old age.

The arterial supply of the mammae is derived from the thoracic branches of the axillary artery, namely, the intercostals and internal mammary. The venous drain is accomplished by means of a fine and complete anastomotic circle around the base of the nipple (called, by Haller, the *circulus venosus*), from which larger branches carry the blood to the circumference of the gland ending in the axillary and internal mammary veins.

The lymphatics, for the most part, course along the lower border of the pectoralis major muscle to the axillary glands. However, some

few lymphatics run along beneath the posterior pectoral and antero-lateral thoracic fascia, perforating the intercostal spaces, and empty into the anterior mediastinal glands. These venous and lymphatic drains are chiefly responsible for all progressiveness of malignancy from the subareolar tissue of the breast.

The nerve-supply of the mammary glands comes from the anterior and lateral cutaneous nerves of the thorax. It is not of material importance when operating; in fact, what nerve branches are encountered may be cut or pushed aside at random.

#### HISTOLOGY

From a surgical point of view the histology of the mammary gland is of extreme importance. The normal make-up of this gland governs every act of the surgeon in making a diagnosis of a pathological condition. From the time of inspection at the office until the patient is removed from the operating table a clear and concise understanding of the normal formation of this gland is very important. The mammæ, being cutaneous glandular structures, compel the surgeon to concentrate his mind upon surface conditions in an early case, and it is always in the early stage that he desires his patients to come to him.

The mammary structures are all derived from the basal layers of the epidermis. From birth to the age of puberty these organs are in a state of constant growth. At an early date they are surrounded by a sheath of connective-tissue. However, at this stage the alveoli, which have developed in the meantime, remain solid and relatively small. In the female the mammary glands do not reach their full stage of development until the last months of pregnancy. They become functionally active at parturition.

*Structure of the Fully-Developed Mammary Gland.*—Each gland is made up of twenty lobes, separated from each other by connective-tissue septa. These lobular divisions are again subdivided, the subdivisions being composed of numerous irregularly round, oval, or tubular alveoli. The alveoli are supplied with minute excretory passages. These unite to form the smaller ducts, which in turn meet and make up the larger ducts. Thus we have a true racemose gland, *likened to a bunch of grapes*. At the surface of the mammilla each



layer duct widens into a vesicle called the *sinus lactiferus*. The number of larger ducts corresponds to the larger lobes, namely, twenty in number. They are lined by simple, cubical epithelium, except near their termination in the nipple, where stratified pavement epithelium replaces the cubical type. These ducts are surrounded by a sheath of fibrous tissue.

When the gland is at rest the epithelium of the alveoli consists of a single layer of glandular cells, nearly cubical in shape. These cells stain deeply, and the internal surfaces will now and then be found to extend into the lumen. At the time when the gland becomes functional fat-globules make their appearance in the distal ends of the cells. This results in a corresponding symmetrical increase in size.

During milk-secretion the glandular elements become separated and the type of cell altered and flattened in appearance, at the same time having a greater secretory ability. In other words, these new-formed cells, or rather altered old cells, have a much greater working power. In the foramen of the areolar papillæ are found numerous small, smooth muscle-fibres which form circular bands around the excretory glands near their terminals.

Lymphatics are very numerous in the connective-tissue stroma between the lobules. These collect and form larger branches, which empty into the axillary glands. Thus it is seen that any stimulus in the connective-tissue will early affect the lymphatics.

#### PHYSIOLOGY

The increase in size of the mammary glands at puberty is brought about principally by the increase in connective-tissue. True glandular tissue remains rudimentary and functionless. However, at the time of conception this glandular tissue is in some way stimulated to growth.

Actively secreting alveoli are formed, and during the last weeks of pregnancy they produce a secretion in elements of true maternal milk, scanty in amount, and known as colostrum. Special stimuli again influence these alveoli after delivery. The mammary gland becomes much enlarged and an abundant secretion is formed.

For two days following delivery this increased secretion still has

the characteristics of colostrum. Following this period, however, true milk becomes evident, and from then on it is produced in plentiful amount. During the period of lactation the amount of milk remains high as long as the gland is under the influence of the act of milking. Should a new conception take place during this period of lactation the composition of the milk is altered and secretion usually ceases entirely.

When the act of nursing, for any reason, is abandoned entirely, the glands enter a stage of turgidity. This is followed by retrogressive changes that finally result in the cessation of secretory activity. Thus are produced the various changes which may take place in some cases under various circumstances, and do occur in all cases as a final result.

#### PATHOLOGY

We now come to the real and fundamental points of the subject of this paper: "A Plea for Early Operation and Extensive Dissection." How can we think of proving our position without first qualifying along pathological lines? It is the intrinsic study of pathological conditions that makes a true and helpful surgeon. We might easily make our subject, "A Plea for Early Pathological Diagnosis." In fact, without this we are wandering in doubt. Let us all, as surgeons, early become energetic students of pathology.

Malignant growths of the mammary gland are classified under two great divisions, namely, *sarcoma* and *carcinoma*.

**SARCOMA.**—This type of malignant growth constitutes about eight per cent. of the cases observed. Possibly the small round-cell type is the more common. It generally occurs early in life, before the age of thirty, although this is not always true. It is very rapid in growth, producing very early ulceration of the skin covering. The entire breast soon becomes involved, giving a uniformly firm feeling. The lymphatic structures are not early involved, for which we have reason to be thankful. However, when this chain of glands has picked up the growth it seems to nourish the original growth in the breast to such an extent that an enormous size is soon attained.

**CARCINOMA.**—A broad division of this variety would lead us to the scirrhus, or hard, and medullary, or softer, tumor. We also might bring into our differential field the colloid variety. However,

this latter is very hard to diagnose before operation, and is at the same time infrequent.

The medullary type seems to come earlier in life than the hard or scirrhus form. As a line of separation it might be stated that the former is found before the age of fifty is reached, while the latter usually comes nearer the three-score limit.

Carcinoma affects those who have nursed children or those who have had some form of mastitis. In making an ante-operative diagnosis these points should always be borne in mind. Trauma very often gives as a final result the carcinomatous variety.

A small nodule may be the forerunner of trouble and will usually appear in one of the inner quadrants of the breast. Early involvement of the ducts brings about a quick fixation and induration around the nipple. The acini growth does not become fixed as early, although it is the more common type found in women. The former will at times be seen in male subjects. Carcinomatous nodules usually occur singly. These growths progress far more rapidly than benign tumors. So far as carcinoma is concerned, we find that the medullary form grows much more rapidly than the scirrhus.

*Small Round-Cell Sarcoma.*—All sarcomata are principally composed of embryonic connective-tissue. This form of malignant growth enters into the class of undeveloped structures. Any form of sarcoma may develop in previously normal tissue belonging to the connective-tissue group. We therefore find that the skin, subcutaneous tissue, intermuscular connective-tissue, and connective-tissue of the glandular elements become early involved. Any form of sarcoma may develop upon preëxisting connective-tissue tumors, such as fibroma, myoma, chondroma, hypertrophic lymphangioma, etc.

When we specially consider the small round-cell variety we at once deal with a very soft and quickly-growing tumor. It develops in the connective-tissue of the motor apparatus as well as the supporting frame-work. We also find these tumors in the skin, testicles, ovaries, and lymph-glands.

On section the small round-cell sarcomata appear milky-white, with here and there a caseous or softened area. When the edge of the knife is scraped across the newly-cut surface a milky fluid is left on the face of the blade.

Microscopically we find that the structure of this type of sarcoma is very simple. The tumor is composed almost entirely of round cells and blood-vessels, with here and there a group of fibrous tissue or a gland. The cells appear very small and frail, possessing a small amount of protoplasm, but have rather large, oval or spherical nuclei, which seem to be highly developed.

Between the cells there is seen a scanty amount of fibrogranular intercellular substance. The blood-vessels of these tumors, as a rule, have very thin walls. Here and there a lymphoid cell may be seen.

*Scirrhus Carcinoma.*—Any carcinoma receives its name from the character of the tissue from which it springs. Thus in the scirrhus variety we have a predominance of connective-tissue, with relatively few cancer-cells. It is this overgrowth of connective-tissue which gives to this tumor its hard, nodular feeling, and therewith its name.

The cell-nests are from the beginning relatively few and small, while the connective-tissue stroma is abundant and hard. Such a tumor is formed especially when the epithelial proliferation infiltrates hard connective-tissue. This formation is most marked in the mammary gland as well as in the skin.

Gradually we find that there is a progressive destruction of a large proportion of the epithelial nests of cells, while at the same time the connective-tissue increases in a very appreciable amount. For this reason a tumor may be soft at the beginning, but later become very hard.

*Medullary Carcinoma.*—Here we are dealing with a malignant growth whose feeling and physical nature are diametrically opposite to those of the previous variety. We find abundant development of cell-nests enclosed by a very delicate connective-tissue stroma. In other words, we have a most active proliferation of the epithelial cells, finally leading to a formation of compact cell-nests. Many of these possess no lumen, since the compaction is so great. The stroma is so poorly developed that necessarily the tumor feels very soft.

**DIAGNOSIS.**—The surgeon who has developed himself along clearly-cut pathological lines finds but little difficulty in making a fairly accurate diagnosis of most breast-tumors with which he deals. In fact, many men are able to make a very creditable assumption



of the pathological make-up of the average breast-tumor which they may meet. It is only fair to state that this assumed pathological condition is very often corroborated by the findings of the pathologist. However, the necessity of determining the condition of abnormal breasts from this standpoint is becoming less frequent as time goes by. We as surgeons do not want to make a pathological *hint* diagnosis on any case which is so far advanced. We desire to meet our patients earlier in their troubles.

Any surgeon should consider it great foresight on the part of the patients if they will come to see him at the time when any abnormal growth in the breast has begun. He should welcome them most heartily when the tumor is possibly no larger than the head of a large pin or even a pimple under the skin. In other words, it is the precancerous stage in which he wants to see his patients, and then he can cure them to remain cured.

Every surgeon should teach his female patients the art of watchful care of the breasts. This should be a part of our regular curriculum professionally, and then a word of caution will later be appreciated. See the breast in the precancerous stage and insist upon complete removal of this gland and a complete dissection of all adjacent structures.

It is nothing short of crime to allow any patient to remain under our watchful eye until an absolute diagnosis can be made from the conditions of the skin, glands, and lymphatic involvement. Many times then we are too late, and the best that can be done will not prevent recurrence. To be sure, such a case must not be given up as hopeless, because life can be lengthened and comfort instituted, but why delay until such a stage?

What we desire most is to cure the condition and not have to meet our patients again within a space of from six months to two years with a recurrence. Of course, many times the patient will not see her physician until the advanced stage of the disease is reached. Here again I would lay emphasis on the fact that we are under obligation to inform our patients ahead of time whenever possible.

Too many family physicians leave any minute growth in the breast and wait for developments. In the meantime the gland is massaged



by the friction of the clothing, as, for example, the corset. Repeated examinations do not help, but, on the contrary, hurt the condition. Traumatism, slight or severe, is most dangerous to any growth of the mammary gland. We have proven through the pathologist that trauma is one of the most frequent soils from which malignant growths of the breast may start.

We have now come to realize that cancer of the breast is not of microörganismal origin. We have come to learn that we are dealing with a cell proliferation, and in every one of these minute bodies we have the fundamentals of true life, so far as the physiological make-up is concerned. Upon this single known fact rests the ability of every surgeon to cure his patient of any malignant growth of the breast. Again, emphasis must be given to the statement, "Operate in the precancerous stage and save your patient from the horrible pangs of cancerous growth of the mammary gland."

It is the desire of every physician and surgeon to shorten the suffering of the unfortunate and at the same time lengthen life most appreciably. The writer dares to state that in the near future every thorough surgeon will have the pathologist examine small segments of mammary gland tissue at various stages during the cycle of life. This is prophylaxis in surgery, and the time will come when we will regard this step equally as serious as we do the administration of diphtheritic or typhoid antitoxin. Most hospitals now require the pupil nurses to receive prophylactic doses of antityphoid serum as a regular routine when typhoid cases are being treated by these nurses. To-day it would be considered a crime if diphtheritic antitoxin were not given as a prophylactic measure when indicated. At the Children's Hospital in Boston, Massachusetts, this antitoxin is given to each patient once every three weeks while they are in the hospital. The records of the hospital will show that diphtheria is practically unknown among the little patients. Such a measure calls for nothing short of applause.

The writer believes that the pathologist will come to study breast-tissue at each semidecade in life after the twentieth year. He will learn the normal variations in mammary gland tissue at these various stages, and will come to the stage when cancerous development can be foreseen.

Before any cancerous growth can make a seeding it must have

the proper soil. It is on the minute characteristics of this malignant producing soil that we, as surgeons, desire the pathologist to give us a report. Let the pathologist study more in detail the particular minute changes which must occur in the mammary gland tissue precancerously. The time is near at hand when he will hunt for the most minute change or for the development of a single cell where it does not belong. He will assume the same position that a bacteriologist now takes when he examines a sputum for tubercle bacilli. To make a positive diagnosis the bacteriologist need find but one tubercle bacillus. This is sufficient ground upon which to make a positive diagnosis of the condition. Just so the pathologist must learn to recognize that particular tissue soil, produced by abnormal change, no matter how minute, in structure, in which mammary malignancy can thrive. He is dealing with a minute cellular formation which can progress only in certain structures and under most favorable circumstances. This strenuous hunt for that minute change or the single cell beginning will place the surgeon in a position where he is able to cure the malignancy and to have it remain cured.

Under these conditions metastasis, *per se*, will become a thing of the past and something to be looked upon as shameful and unworthy of good, clean surgery. Secure the mammary gland in the precancerous stage and then amputation, accompanied by the thorough dissection as outlined by such men as Hawley, Halsted, Rodman, and Jackson, will allow the surgeon to cure that most dreaded condition, malignancy of the breast.

The writer also dares to state that the immediate tissue of the breast will not in the future be considered as positive evidence that malignancy is present or absent. Possibly a small nodule may appear in breast-tissue and microscopical examination may show this to be simple inflammatory tissue. Such a condition can readily be assumed to spring from the indirect results of a more severe process located within the deeper tissue of the mammary gland.

In the future we will find ourselves dissecting a simple lymphatic gland or a small group of the same located in that quadrant bounded by the upper mammary border below, the clavicle above, the sternum internally, and the axilla externally. The make-up and contents of one or more of these glands as shown by the microscope will bring to the surface what may be developing within the deeper mammary

structures. Such a measure would be, in the majority of cases, prophylactic, and here is where the surgeon should concentrate his mind when dealing with the breast.

Our patients will need to be educated to this method of prevention, yet how willingly they will submit when the matter is explained! One word of caution might be mentioned in passing. No surgeon has a right to consider simple inflammatory nodules in the breast in any other light than that such may mature into malignancy if allowed to remain. This is especially true when such a nodule persists or leaves any indurated portion behind. I would not have the reader get the idea that the writer would amputate all breasts which may contain a simple, traumatized area, of known cause, that may succumb to proper care and precautions. Such a case should not be allowed to go on unwatched, however, as any nodular condition of the breast should be considered malignant at all times until proved otherwise.

Outside of an acute mastitis, any inflammatory area or dull induration which persists as such for more than two weeks can show nothing but the most complete surgical interference. We may see the near future produce, in the field of chemical pathology, most satisfactory prophylaxis, so far as the breast is concerned. That brilliant work of Herter's dealing with this side of science will be an incentive for deeper work along these lines. The writer believes that in this field alone it is possible to develop some means forwarding prophylactic measures in breast surgery. If such can be attained, the complete development of *any* malignancy will be a thing of ancient history in breast surgery. What a field to be developed and what a result to suffering mankind!

When breast malignancy has become established we find ourselves dealing with a tumor which, when seen in the early stages, is hard and somewhat movable. Later this tumor becomes adherent to the skin, most markedly about the nipple. A further stage sees this adherency extending to the underlying pectoral muscle. The degree of hardness of any breast tumor, of course, depends upon its pathological make-up. Nevertheless, all varieties are pronouncedly firmer than any portion of a normal breast. At first the tumor is rounded and can be easily outlined, but later it becomes more diffuse and nodular.

When examining the nipple we may find that it has become fixed

or possibly retracted. Early ulceration may take place at this point. At times pain is an early and prominent symptom, yet no special stress should be laid on the fact that pain is absent. Upon palpating the axilla we may find that the lymphatics can be easily outlined, singly, grouped, or in chains. Even the supraclavicular lymph-nodes may be felt when the condition is more advanced. These parts should always be carefully examined, not once, but on successive days or at intervals of only a few days.

In every case where there is a question of malignancy of the mammary gland the liver should be very carefully percussed and palpated. The inguinal glands should be very carefully palpated and the general physique noted. No short-cut route should ever be used in examining any patient, as thoroughness is the keynote to success.

Some patients may show certain complications, such as cough and difficulty in breathing. These symptoms will at once draw attention to the mediastinal glands as possibly having become involved. Eisen-drath has mentioned, in his thorough and most helpful work, that he terms "cancer en cuirasse." Here we find the skin over the entire front of the chest has a board-like consistency, the "armor-like cancer." We may even have the metastases to the lung, pleura, and brain.

Bone involvement as a chemical metastatic complication of breast malignancy should not be overlooked. It is a known fact that chemical deposits in the osseous structures are altered many times, and we find fractures occurring, following a most trifling injury or even at times spontaneously. This is probably not due to direct metastasis, but rather to altered trophic conditions belonging in the realm of chemical pathology. Trophic changes may become so marked as to produce osseous overgrowths in the skull and spine. Such conditions should always be sought for and the Röntgen ray brought into use whenever possible.

One important sign may at times be overlooked, viz., the œdema of the hand of the involved side. This œdema may not be apparent at first, and pressure pitting may not be present at all, either in the hand or forearm. However, questioning may elicit the fact that the patient for some time has noticed a thick, leathery feeling in the hand. The fingers seem more plump than those of the opposite hand. The writer has also noticed one sign which he has never found men-



tioned in other works. Whenever œdema is present in the hand and forearm both arms should be compared while hanging naturally at the sides. If the upper arms are now very carefully compared it will be noted that on the affected side the biceps muscle seems to be rotated outward on its long axis. This sign will not be noted except under careful scrutiny. The first appearance will make it seem as if the upper arm of the affected side was enlarged. However, if the circumference measurements are taken they will be found normal. The actual condition is an external rotation of the biceps, this being pushed outward by the pressure of the axillary glands upon the subcutaneous fascia. The author has found this sign very persistent when the axillary space is diminished by glandular enlargements or other growths. It seems worthy of note.

The résumé under the heading "Diagnosis" brings forward many conditions which it is hoped promptness on the part of both patient and surgeon in the future will make as infrequent as the monster ovary cysts of a decade or more ago. It seems feasible to assume that proper office talks along educational lines will bring our patients under our care in the precancerous stage. Let us hope we may see this progress within the present decade.

#### PROGNOSIS

Every surgeon should hope for that day when we can state to our patients that proper surgical interference will result in absolute cure of this dreaded condition. Prognosis will then be the vital issue to the patient, but a matter of little import to the surgeon. When we feel that we have trapped the disease in the incipient stage we at once become so concerned in the matter that nothing but success will result. Get the cases in the precancerous stage and the path to a good prognosis is smooth. Procrastination means failure. There should be no division in opinion on this point among our profession.

In the writer's clinic there was a case of recent date which gave a history of two months' duration. This woman had an easily palpable tumor in one of her breasts. Pain was a most prominent symptom, and the local condition led to one of general nervous upset and worry. After proper examination and a week of observation,



during which time I saw her three or four times, I explained her condition in detail and advised surgical procedure.

This patient hesitated and in due time visited her old family physician, who was located in a town somewhat distant. This physician made an examination and found the then freely movable breast-tumor and no glandular involvement. He advised delay and further observation. In the course of a few days she again returned to me in a very nervous state. I asked for a consultation, which was granted. As a result an operation was advised at once. She consented and the Jackson operation was performed, with complete dissection and the most satisfactory recovery.

I cite this case to show how professional opinion may differ. Why should such be the case at this age of progressive surgery? What else can be done in a case of this type but an early and complete operation? Every physician should become aware of the dangers of delay and refer his cases to a surgeon at once. When such complete coöperation can be maintained we shall always expect to give a most favorable prognosis in the early cases.

#### RÉSUMÉ

In reviewing the above-mentioned facts it seems to the writer that the complete wide dissection of the breast entire and a careful picking up of all adjacent lymphatics are the important points. To remove the tumor by a wide incision, such as Jackson and Rodman have so ably outlined, is the proper move in modern surgery.

The beginning should always be at the top in the clavicular and axillary regions. Never leave a doubtful berry of fat, as this nodular adipose may be a gland. The day of postoperative pressure up the axillary venous system has passed in modern surgical procedure, so far as the breast is concerned. Remove both pectoral muscles of the affected side and see that the fascia of these muscles is carefully scaled off.

We know to-day that the fascia and muscles of this region are ground-gainers for the lymphatic spread of the disease. When necessary we must go well down to the trapezius muscle, and lay bare the strong and wide latissimus dorsi. The lymphatics lying upon these muscles and intermingled with their fascia converge to the axillary

glands. What merciful work is already accomplished when the pectoral muscles and fascia have become lifted away, thus blocking upward lymphatic flow!

We should always realize that a certain few of the lymphatics of this region pass through the intercostal spaces and join the anterior mediastinal glands. Even diaphragmatic glands are intercommunicable. What a risk there is in failing to carry out thorough dissection of all superficial and some deeper, extrathoracic glands! Chain upon chain of these disease-spreading channels must be extirpated and the distal avenues ligated.

Every breast amputation should be accompanied by such an extensive dissection as will reach well down upon the rectus abdominis and even the upper inner angle of the external oblique muscle. The sheath of the former should be scaled off, in part at least, and possibly a portion of the aponeurosis of the latter. Endeavor to so constrict all lymphatic avenues that the lymphatic duct or thoracic duct, depending upon the side affected, shall not drain even one cell of the diseased tissue. Remove the necessary muscular tissue and scale off the fascia well, as this procedure means a lower percentage of recurrences.

When we consider how open the mammary gland is to traumatism and local irritation, great caution should be taken to prevent this taking place along the linear scar. Postoperatively protect this scar by soft pads and make it comfortable for the patient. Not only can malignant disease spread by lymphatics, but also by the veins. We do not need to consider the arterial system in this ill category.

Remember the histological make-up of the breast. The cubical epithelium lines the deeper portion, and it is this form which will take on malignancy much earlier than will the more superficial stratified, pavement variety. We must also bear in mind that the normal physiological changes of the various decades of life may cause very rapid alterations in tissue type.

The older idea of placing importance upon which breast quadrant the tumor is in should be put aside. Any tumor is dangerous, irrespective of quadrant. Consider the breast as a whole and deal with it as such. We must also never forget that all benign growths may become malignant sooner or later. This is merely a stepping-stone for further and more serious trouble.

## CONCLUSIONS

1. The physician should begin to teach his patients the dangers emanating from neglecting to have the breasts examined regularly after the twenty-fifth year.

2. Every surgeon should have at his tongue's end the minute anatomy, the definite physiology, a clear picture of histology, and the important pathology of this part of the human system.

3. The lymphatic system should be considered an avenue for the spread of malignancy of the breast, as well as a bulwark for defence. Every chain of glands leading from the mammary gland should be regularly and carefully watched.

4. The variations in the adolescent and fully-developed breast must always be kept in mind.

5. Every surgeon should group the various malignant growths of this part of the body so that he may recall this classification at any necessary moment.

6. The pathologist should be consulted frequently in every case.

7. In making a diagnosis the physician should be free from too marked conservatism, if the outcome is to be satisfactory to all parties concerned. Give malignancy the benefit of a doubt.

8. It remains for the surgeon to keep the pathologist and researcher ever alert and ready to bring forward some new idea for aid to better early, definite diagnosis.

9. Outside of acute mastitis, any inflammatory area or dull induration of the breast should be considered malignant or that it may become such at an early date.

10. Ever be on the watch for complications of breast malignancy. No one desires to see the disease reach this stage, and this is just what early radical treatment prevents.

11. Let us urge our brother physicians to send these questionable breast cases to the surgeon in the precancerous stage.

12. Early operation accompanied by extensive dissection is the only clew to complete eradication of the trouble. Patients desire to block recurrence, and it is our duty to serve all patients extremely well.

13. Every complete operation should have for its aim the complete removal of lymphatics, muscle, and fascia, careful ligation of the

dreaded venous channels, and prevention of hemorrhage, which expedient and accurate work will accomplish.

14. Extended observation, conservatism, and an absolutely sure diagnosis in every case should not be tolerated in breast surgery.

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# LYMPHATIC ŒDEMA: ELEPHANTIASIS: OBSERVATIONS AND REMARKS, WITH REPORT OF OPERATIONS

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THE influence of *Filaria bancrofti* as the causative factor in elephantiasis has recently been given much attention, but there can be little doubt that many cases diagnosed as this disease, especially among those occurring in temperate climates, are the result of pathological changes that produce lymphatic dilatation and obstruction. A serious neglected appendicitis, scarlet fever, typhoid fever, cirrhosis of the liver and kidneys, traumatisms, operations involving the inguinal and pelvic regions, phlebitis, carcinoma, etc., may result in an œdematous condition that would be diagnosed as elephantiasis. We have, then, two conditions which must be carefully considered in making a diagnosis, *i.e.*, non-filarial or lymphatic œdema and true elephantiasis. The gross (macroscopic) appearance and the objective symptoms cannot be relied upon as sufficiently accurate to always form a correct diagnosis, and even with a careful histologic study cases seen late in the disease must at times remain unclassified.

In the presentation of the subject of this paper I shall report some of my surgical cases, but I trust not in tiresome detail.

CASE I.—Mrs. A. B., æt. 40, with good family history, came under my observation in May, 1866. I had seen somewhat similar cases in the hospitals, but this, a private patient, gave me an opportunity for careful study. She was the mother of three children, and had passed through each confinement without any increase of her trouble. At about the age of twenty she had noticed an enlargement of the leg below the knee, which gradually increased and involved the thigh. No acute symptoms had presented at any time, there had been no traumatisms, nor could pressure upon vessels above be discovered, from growths within the abdomen, except as to the possibility of an attack of appendicitis—inflammation of the bowels—which she had had when a child. The etiological factors being so obscure, the diagnosis of elephantiasis was made. She was a native of the interior part of New York State and had not lived elsewhere. The case remained under observation for a number of years. Careful attention was given to her general health, as much rest as



possible advised, and pressure by means of bandages was applied from the toes up to the groin. This afforded relief, but when removed the general œdema would return. Aside from the weight of the limb and the rather increased tired feeling resulting from doing her own housework no other marked symptoms presented. There was no ulceration at any time. Patient jokingly remarked that "if the limb grew too large she would permit an amputation."

The case was evidently one of lymphatic œdema, possibly congenital in character. No other like conditions presented elsewhere about her system. The limb became larger later in life and gave her much weariness, but operative surgical intervention was not attempted at any time.

Since observing this patient I have seen a few cases of distinct lymphatic œdema of the lower extremities; in one of these there was enlargement of the scrotum and in another of the vulva. I have also seen several arm cases, following removal of the breast in the female, and one case of œdema of the arm in a young woman without tumor or operation. I have had no experience of cases of cervical rib causing swelling of the arm, as reported by Osler.

The study of the pathology and treatment of these cases has been exceedingly interesting. I have met with a number of cases of lymphatic œdema, appearing both early and very late, in operations for removal of carcinoma of the breast. It has been my experience that enlargement of the arm frequently occurs after extirpation of the breast, the chest muscles, the thorough removal of the lymph-glands, or even where a less severe operation has been performed. The œdema of one or both arms that presents within six months or a year after operation will in most cases yield to the application of bandages, from the fingers up; an elastic bandage may also at times be used with beneficial results. Where there is an attendant neuritis great comfort may sometimes be afforded by the use of the galvanic current, and also by the employment of the X-rays. Many of these cases are doubtless the result of blocking of the lymph channels. I have been greatly impressed by their persistency, and in contrasting them with those cases in which a portion of the axillary vein had been removed I have noted that the œdema in these latter cases was not so persistent and yielded more readily to treatment, the lymph channels, evidently, not having been so seriously involved. Cases of this kind have been designated by many surgical writers as elephantiasis.

CASE II.—Mrs. C. D., æt. 72. One sister dead—due to a recurrence of a tumor of the neck believed to be a carcinoma. Patient menstruated at 13; one child; at the age of 50 had excessive flow for several months, just at time of menopause. Latter part of 1904 noticed a lump in left breast, and came under my observation in December, 1905. Diagnosis, adenocarcinoma. Operation, removal of tumor, together with axillary glands, February 12, 1906. Wound healed kindly. Following six months there was some œdema of the left arm which yielded to bandaging. Health remained excellent for over two years. Patient remarkably active and free from pain. During October, 1908, she noticed a growth in right breast, not painful, but with some retraction of the nipple, and her previous history aroused suspicion as to the possibility of its being malignant. Not improving under treatment, she was admitted to the hospital November 8, 1908, and operation done for complete removal of the breast, together with the axillary glands. In view of her age and the left breast having behaved so well it was thought best not to perform a more severe operation. The wound healed quickly, and the patient left the hospital in less than three weeks. She made a good recovery, but in May, 1909, noticed some pain in the cicatrix of the right breast, and a nodule could be felt. She was admitted to the hospital June 14, 1909, and the old scar-tissue, together with the pectoral muscles and subclavian glands, removed. Wound healed kindly and in ten days she returned to her home, remaining well until August, 1910, when lymphatic œdema of the right arm presented in a most marked manner. Soon after an enlargement could be felt just under the right clavicle, evidently involving the periosteum. Bandaging of the arm was carried out fairly successfully and reduction of the enlargement followed. X-ray treatment relieved pain and there was some lessening of the metastasis that involved the second and third ribs. There was no ulceration at any time, but patient gradually failed, there was metastasis to the stomach, and she died June, 1911.

This patient had been kept pretty thoroughly upon arsenic and iron from the time of her first operation, and the bandaging did have an excellent effect in relieving the œdema.

CASE III.—Mrs. E. F. Operated upon May, 1889; entire removal of left breast, with lymphatic glands. About two years previously had been treated by Dr. T. Gaillard Thomas, of New York, for profuse uterine hemorrhage. No surgical intervention, and, after a few months, menstruation became normal and patient showed no further pelvic symptoms. She remained in very good health until about November, 1909, when she noticed a swelling of the left arm, which gradually developed into marked lymphatic œdema, extending from the finger-tips to the shoulder. No evidence of any return of the disease could be noticed at this time. She had a decided arteriosclerosis and was benefited by the use of nitroglycerin. Patient gradually developed a neuritis of left shoulder and arm, and it was thought there was some enlargement of the supraclavicular glands, but the swelling of the arm did not increase. The œdema of the arm and the neuritis continued, however, at times better, then again more severe, and she was placed under the care of Dr. E. A. Bartlett, of Albany, New York, July 12, 1910, who has very recently, July, 1911, given me the following report of her case:

"Condition of patient upon coming under my observation: Left arm markedly œdematous from shoulder to finger-tips, voluntary motion in arm and hand lost, complete anæsthesia to touch, heat, and pin-prick over whole of posterior aspect

of arm, forearm, little finger, and ring finger. Extensors over same area give no response to any form of electrical stimulation. Extensors of thumb, index and middle finger, as well as all of the flexors, respond to faradism (4 in. C. D.). Dull "booming" sensation complained of in arm and forearm and "aching" pain under left scapula; otherwise no pain. There is a tumor, 1 in. by  $\frac{3}{4}$  in., appearing above left clavicle and lying just back of posterior border of sternocleidomastoid muscle.

"Diagnosis: Compression of brachial plexus by tumor described; tumor presumably malignant.

"Treatment: Röntgen ray to tumor; high-frequency current through vacuum electrode, galvanism and faradism, as from time to time indicated, to arm, forearm, and hand. Medical treatment left to my colleague.

"Present condition: Tumor in neck has disappeared from above clavicle; œdema slightly decreased; pain under scapula gone; pain in arm lessened and index finger and thumb can be approximated; other motion not restored.

"Three months ago a tumor, 1 in. by  $\frac{3}{4}$  in., appeared on right side of neck at upper posterior border of sternocleidomastoid muscle which, under Röntgenization, has been reduced one-third. This tumor is also probably malignant."

This case is somewhat rare because of the very late appearance of the return of the carcinoma. The lymphatic œdema of the whole arm was very marked, but yielded most decidedly to the use of bandaging and electrical treatment. However, the patient has the evidence of carcinoma of the glands of the neck, and while being benefited by treatment the case is very serious.

Ligation of the artery for relief of lymphatic œdema of the lower extremities does not present a record of entire satisfaction as regards results. There has been a well-marked mortality list, due to gangrene following operation, and, even though this last serious complication does not present, the results are not always in the line of cures.

CASE IV.—For some years I was much interested in watching the case of Mr. G. H., æt. 50, German, porter by occupation, who had been under my observation in the dispensary and hospital for a period of fifteen years. For some twenty years he had noticed a gradual enlargement of the right leg until it presented the characteristic appearance of lymphatic œdema, with a tendency to ulceration; the ulcers at times healed under continuous treatment by bandaging, yet the limb slowly became less useful and his symptoms more serious. He had been admitted to the Albany Hospital at various times, experiencing marked relief by rest, elevation of the leg, and the use of bandages. In December, 1907, Dr. Willis G. Macdonald and myself performed ligation of the femoral artery, in Scarpa's space. The patient made an excellent recovery, but there was not much diminution in size and little permanent improvement. A year later the ulcers appeared in a more serious form and amputation was done through the middle third of the thigh. The patient convalesced nicely and was very much more comfortable afterwards, but in this case ligation of the artery certainly did not bring the desired relief.

CASE V.—Mr. I. J. In many respects this case seems quite unusual, and leads to the study of the possibility of phlebitis, having produced lymphatic œdema which many years afterward resulted in malignant growth. This patient was a native of Canada, and by occupation a carpenter. He served as a private during the Civil War. In the early part of his service he passed through an attack of typhoid fever, complicated with phlebitis of the right leg. Later he suffered considerable distress, after a long march, and the limb began to enlarge, increasing slowly for a period of twenty-five years, but not until about 1885 did he suffer any great pain. At this time there was occasional sharp pain and the limb showed a more decided tendency to enlarge about the knee. He entered my service at the Albany Hospital June 6, 1888. There was considerable œdema of the leg itself. From the toes up to the upper third, and from there up to the groin, there was a uniform enlargement more markedly distinct about and above the knee, and on the inner side producing a nodulated induration at first thought to be the remnants of his old venous obstruction. His thigh measured 30 in. in circumference. Bandaging from the toes up to the knee, and rest in bed, reduced the size of the limb at that point, but he could not tolerate bandaging from the knee up to the groin. The patient was unable to go on with his occupation, he was losing somewhat in flesh, was more or less restless at night, and no improvement following treatment, and, considering the case not proper for ligation of the femoral artery, amputation was suggested. To this he consented, after a period of palliative treatment. Operation was done in the upper third of the thigh and patient made a good recovery. An examination of the specimen removed at the operation (not having, however, the benefit of an expert laboratory pathologist) showed the bone free from infection, but the growth about the knee, and extending up one-third of the thigh, seemed to be of the nature of a distinct enchondroma. The patient did well and was alive ten years later.

At the present time a more reliable report could have been procured regarding the possible malignancy of this case. Did we have here a distinct case of lymphatic obstruction in which there was later grafted on a malignant condition?

I have had two cases, male and female, of what I believe to have been hæmatochyluria associated with chylous dropsy. The male was operated upon for supposed tuberculous peritonitis and recovered. In the case of the female, at the time supposed to be one of ovarian tumor, operation plainly revealed chylous dropsy, the patient dying later of peritonitis.

A more correct diagnosis could be reached at the present time by aid of careful laboratory investigation.

CASE VI.—Mrs. K. L., æt. 38, admitted to Albany Hospital May 4, 1911; discharged May 29, 1911.

Diagnosis: Elephantiasis, right leg, non-filarial.

Treatment: Operation; wedge-shaped pieces removed.



Personal history: Patient married at eighteen; mother of two boys, alive and well. Complaints of "enlarged leg."

Family history: Excellent. No history of tuberculosis, cancer, rheumatism, heart or kidney trouble.

Past history: Patient had measles and mumps; never had typhoid fever, pneumonia, or suffered any traumatism. Menstruated at fifteen; painful and regular; normal in amount and lasting 3 to 5 days. Bowels constipated; appetite good; weighs about 140 pounds, 125 when married.

Present trouble: Began about fourteen years ago with sharp pains in limb above knee. Somewhat swollen at the time, but did not begin to enlarge very much until five years ago. Patient thinks it began to enlarge all over, not any particular region enlarging more than others. Does all of her own housework, which tires her considerably, and she becomes very nervous.

Measurements: Around foot  $9\frac{1}{2}$  in., around ankle  $18\frac{1}{2}$  in., below knee 15 in., around knee 16 in., above knee  $17\frac{1}{2}$  in., middle thigh  $21\frac{1}{2}$  in., top of thigh  $23\frac{1}{2}$  in.

This case was carefully studied by Dr. Ordway, of the Bender Hygienic Laboratory, and his assistants. Several examinations of the blood were made, at various times, during the night, but no *Filaria bancrofti* found.

Operation: Extirpation of three portions of wedge-shaped pieces of tissue, one over anterior aspect of lower third of leg, transversely, just above ankle, and two over posterior lateral aspect, each wedge corresponding to points of greatest redundancy. Very slight bleeding. Tissue blanched and œdematous in appearance, with apparent partial thrombosis of blood vessels. Slight bleeding points tied with cumol catgut ligatures. Wounds closed with silk-worm gut. Two rubber tubes inserted for drainage.

The report on the surgical specimen from Bender Laboratory, received May 8, 1911, from the Albany Hospital, in the case of Mrs. K. L., is as follows:

"*Gross Examination:* The specimens consist of two large elliptical masses of thickened skin and subjacent fat and fibrous tissue; these measure respectively 25 cm. by 4.5 cm., and 23 cm. by 4 cm., and the combined weight is 450 grammes. There are also several smaller fragments of similar tissue weighing 150 grammes.

"The skin is rough and furrowed and has in general a 'puckered' or shrivelled appearance, as if it had contracted after stretching. From its under surface dense fibrous strands invade the subjacent fat. The entire subcutaneous tissue, including the fat and fibrous tissue, is pale and has a general homogeneous, glassy, or 'mucoid' appearance. The fat is of very light yellow color.

"Fragments are removed from various portions of these specimens, fixed in 10 per cent. formalin and Zenker's fluid, embedded in colloidin and paraffin, and stained by the hematoxylin and eosin and eosin-methylene blue methods.

"*Microscopic Examination.*—The subcutaneous lymphatics are abundant and markedly dilated, and all the tissue elements are widely separated by œdema.

"The *epidermis* is in places slightly atrophic and shows moderate keratosis. At the junction of the epidermis and subjacent tissue the former has a toothed or comb-like appearance as if drawn away from the latter. The cells of the sweat glands are swollen and the cytoplasm is pale and granular.

"The *subcutaneous fibrous tissue* shows marked separation of the collagen fibrils by œdema.

"The *intrinsic* smooth muscle fibres of the skin are separated and swollen.



The subcutaneous fat appears small in amount, as the vacuoles are encroached upon by the swelling and fibrillation of the connective tissue between the fat globules.

"There are scattered ill-defined foci of lymphocytes and plasma cells, with occasional polymorphonuclear leucocytes and tissue mast cells. In places these accumulations of cells are distinctly perivascular.

"The smaller blood-vessels and capillaries stand out prominently in contrast to the loose (œdematous) connective tissue surrounding in a way similar to those in the umbilical cord and chorionic villi. The coats of the arteries are thickened, the muscle fibres are swollen and separated; between them are 'vacuole-like' spaces. The adventitia is fibrillated and the intima is irregularly thickened. In places the endothelium is shrunken and shows a varying degree of degeneration. The blood-vessels contain but few corpuscles.

"The lymphatics are abundant and markedly dilated and occasionally there is a break in the continuity of the wall, as if it had ruptured. Many of them are empty, others contain pinkish granular (œdema) and spicule-like material (fibrin), and in still others are lymphoid cells in varying numbers."

Patient made an excellent recovery; however, on the fifth day she had a rise in temperature, and there developed a marked inflammation of the internal saphenous vein which yielded to treatment, causing no serious complication. The absolute rest, elevation of leg, and operation resulted in the limb being nearly the size of the other when she left the hospital. Since then the few reports regarding her case have been favorable.

This case was a typical one in appearance, careful laboratory examination showing it to be non-filarial, and the operation illustrates more than any other point in the study of these cases the comfort that comes to the operating surgeon through having careful bacteriological and pathological tests made by competent experts.

The diagnosis having been made, the treatment of to-day would consist in a thorough examination regarding the possibilities of any organ presenting a pathological condition, or outside etiological factors producing lymphatic changes. Proper attention should be paid to all the functions of the body; the surroundings of the patient; relieving the patient of any malnutrition or imperfect assimilation of food; the use of hot baths; elevation of the limb; the application of bandages; absolute rest, so far as possible; employment of the X-ray, with cinchonization and various forms of electricity; Castellani's treatment by means of thiosinamin (fibrolysin); the operation of lymphangioplasty; sterilized silk thread, subcutaneously employed, and the destruction of fly and mosquito elements that may be present. In the various surgical operations one must pay especial attention to the technique, and avoid infection. The veins as well as arteries

should be carefully ligated, and, as in the case of Mr. K. L., thorough drainage must be provided. The danger of hemorrhage must also be carefully considered.

All therapeutic agents failing, surgical intervention becomes proper, such as ligation of arteries, or incisions that may result in a lymphorrhœa. Longitudinal incision, especially of the arm, in cases of tense, lymphatic œdema is of service, and, finally, the operation of Rogers, of India, for removal of wedge-shaped, elliptical portions of the hypertrophied tissue. This, to the writer, promises better results than more serious surgical intervention in the way of removal of all the tissue, which does not seem to have been very favorable.

## THE VALUE OF EXERCISES IN TREATING CERTAIN CASES OF ACQUIRED INGUINAL HERNIA<sup>1</sup>

BY R. TAIT McKENZIE, B.A., M.D.

Professor of Physical Education and Therapy in the University of Pennsylvania,  
Philadelphia

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My attention was first directed to this subject by the account of his case given me by a surgeon at present practising in Philadelphia. At the age of 18, while hunting, he was thrown against the pommel of his saddle, and shortly afterward a left inguinal hernia developed, becoming very large. He wore no truss, however, and did not limit the activity of his habits, playing football, riding, and running.

Three years later he enlisted for military service in Cuba and was compelled to wear a truss. While in Porto Rico he brought on a right hernia while lifting heavy bales of hay and had to get a double truss, which he wore for about three years, riding, playing football, and engaging in all kinds of athletic sports, during which time the rings gradually closed. He then abandoned the truss, and for the last ten years his abdominal rings have been intact and his general condition excellent.

This extraordinary case made so much impression upon me that I have been observing all cases in the incoming freshman class at the University of Pennsylvania, about 1500, and have had occasion to treat a number of men in middle life, referred to me through the good offices of other surgeons, cases that either should not, or would not, consent to operation, and yet who did not look forward with complaisance to the continuous wearing of a truss. During this time I have seen about 62 cases in which exercise would be indicated, either alone or in conjunction with support.

About 95 per cent. of all hernias are inguinal, and of these 95 per cent. are indirect, and, of course, I will not consider femoral, umbilical, or other forms in which the exit is made through a fibrous opening.

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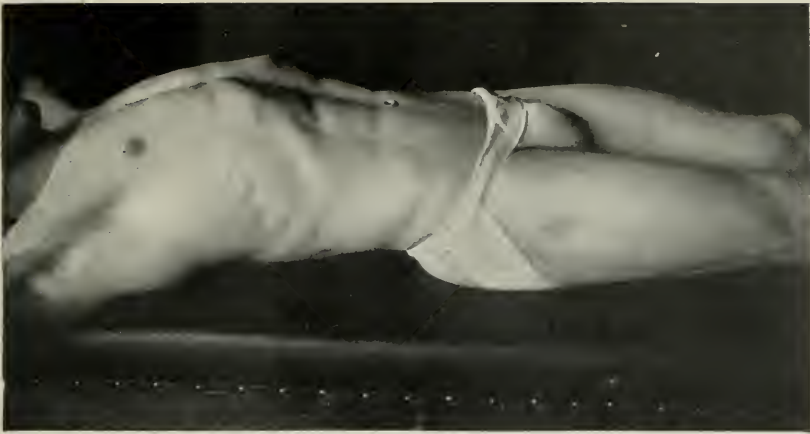
<sup>1</sup> An address before the Philadelphia Academy of Surgery, January 8, 1912.

Inguinal hernia, and especially the indirect form, has to do with rings and a canal that are partly muscular and so are capable of modification by whatever will affect the action and tone of these structures. Looked at from within, the lower part of the anterior wall is divided into three fossæ, the supra-vesical, the inner and the outer inguinal, by the cords formed by the obliterated hypogastric and the patent epigastric arteries, but it is the exception rather than the rule to find a depression or dimple in the peritoneum at the site of the internal ring (Packard); the transversalis fascia, which is perforated by the cord and accompanies it as the infundibuliform fascia, showing no opening or weakness at this point, except in congenital cases, in which the inguinal canal has never closed. The transversalis fascia forms, then, a continuous and fairly uniform sheet, lining the muscle from which it takes its name. This muscle, besides its other origins, has fibres arising from the outer third of Poupart's ligament, and its lower edge shows scattered fibres that fade insensibly into the fascia, the line of demarcation being frequently indistinct and the internal ring being marked only by the *vas deferens* hooking around the epigastric artery. Extending downward and inward, and overlapping the origin of the transversalis, in the internal oblique, its thicker and better organized muscular fibres forming a lid over the internal ring, and its crescentic edge curving downward and inward to join the transversalis in forming the conjoined tendon, whose outer and inferior border is thin and ragged, with ill-defined edge, fading into what is known as the thin spot of the abdominal wall.

Covering them both is the powerful aponeurosis of the external oblique, with its pillars split to allow of the exit of the spermatic cord, but insecurely held together by the scattered intercolumnar fibres, forming the external ring, which varies in its attachments from a scarcely discernible slit to a hole admitting easily two finger-tips.

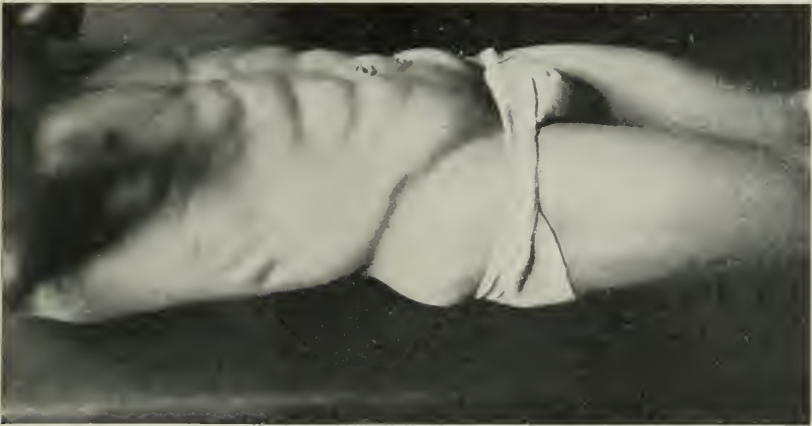
In infancy these inner and outer rings are almost opposite, but as the ilia grow and extend outward and upward the internal ring follows them, and in adult life the canal may be  $1\frac{1}{2}$  inches in length. In any lateral flexion of the trunk where the oblique muscles are in action the whole region is flattened and the pillars of the external

FIG. 1.



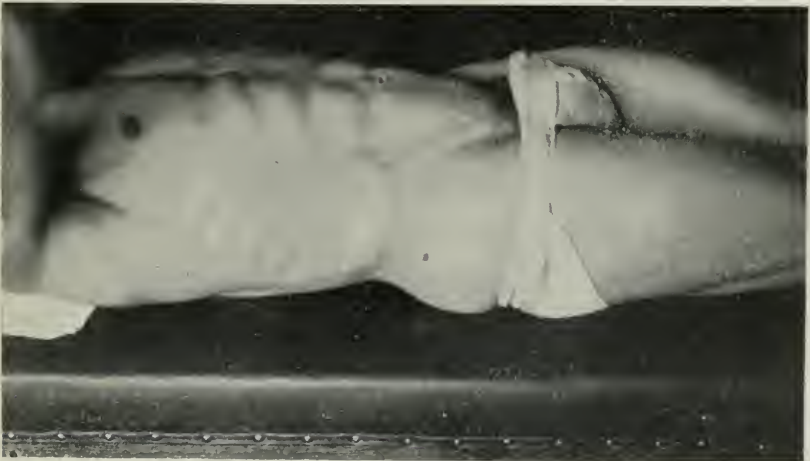
Patient lying with abdominal muscles relaxed.

FIG. 2.



The beginning of flexion of the trunk, showing the recti in powerful contraction with the obliques relaxed, allowing the lateral parts of the lower abdominal zone to bulge forward and distend the rings.

FIG. 3.



The combination of flexion showing the obliques in contraction at this later stage of the movement

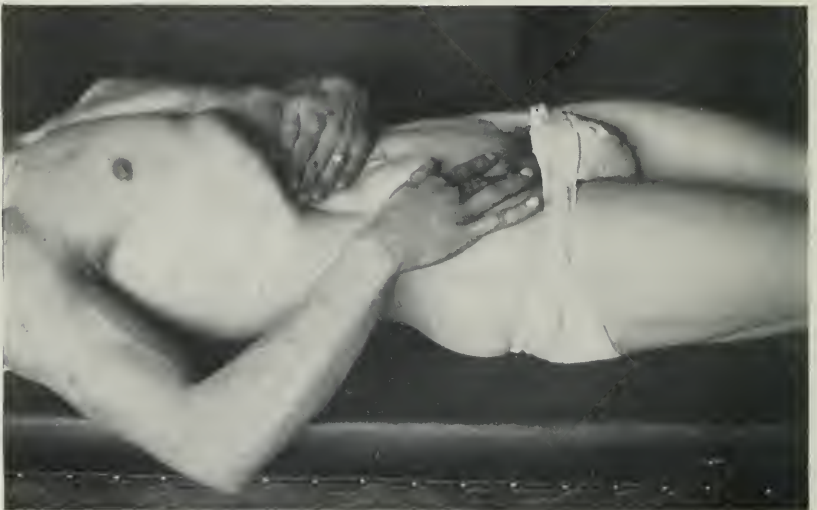


FIG. 4.



Flexion of the trunk and twisting from the affected side, showing the finger protecting the right abdominal rings as in Exercise 3.

FIG. 5.



Abdominal breathing, illustrating method of protecting the right abdominal rings by the second finger of the right hand held flat against the wall, as in Exercise 1.

ring snap together with the first beginning of contraction in the external oblique.

Flexion of the trunk is done mainly by the recti, and, especially at the beginning of the movement, the oblique remain relaxed and the lower inguinal regions tend to bulge symmetrically and evenly. (Fig. 1.)

This general yielding of the region just above Poupart's ligament gives color to the belief that in hernia acquired in adult life or in late childhood there is nothing but the most casual connection with the cord. Some dissections have shown the breach to occur an inch outside the internal ring, and every clinician has experienced the difficulty of determining the exact point of entrance of the hernial sac, especially after the involved structures have been stretched and displaced.

While the point of entrance of an inguinal hernia may vary throughout almost the whole length of Poupart's ligament, its point of exit is always between the pillars of the external oblique, loosely held together by the intercolumnar fibres. When the external oblique muscle is contracted, these pillars are pulled together, as can be demonstrated easily by passing the finger up through the tubular prolongation of the external spermatic fascia and having the patient raise his head and shoulders from the table.

In the movements of straight flexion of the trunk the rectus muscle only is employed at the beginning and the relaxed oblique muscles are distended, forming two distinct pouches or weakened areas over the lower abdomen (Fig. 2), and by the time they contract in self-protection the mischief may have been done.

It is in such conditions and under such circumstances that hernia is likely to be acquired, because hernia, like other swellings, enlarges in the line of least resistance. Perhaps one of the most potent causes is that posture in which the abdomen is protruded and the chest sunken, forcing down the abdominal contents on the relaxed lower zone, and I have been struck with the number of cases in which hernia came on unconsciously without apparent cause, other than perhaps a long walk or a fatiguing day's standing. Even repeated and violent effort seems less fruitful of cases than the dull and steady pressure on the relaxed abdominal walls.

Muscular atony may, of course, follow severe illnesses, especially where there is great loss of fat, but there is also the atony that accompanies sedentary habits, and begins to tell on those approaching middle life, especially choosing the unused abdominal muscles as the place where the deposit of fat will be undisturbed or undermining their powers of contraction, resistance, and control.

In a great number of cases there seems to be plenty of warning in the shape of vague pains in the region, and it is a frequent experience to be consulted for these pains and to find the ring patulous, but showing no definite hernial protrusion. This condition is closely associated with ptosis of other abdominal organs and with other signs of a general ligamentous and muscular relaxation.

Altogether, the impression I have gathered from the cases coming under my observation is that the onset, except in rare cases, is much less dramatic than has generally been thought.

The great majority of cases tend to improve if the hernia can be reduced and kept in place long enough to give the rings time to recover from the stretching to which they have been subjected; if, in addition to this, the oblique muscles be systematically strengthened by accurately applied exercise. To those patients who object to operation for a radical cure exercises may be prescribed, due precautions being observed, which will ensure a cure in about 70 per cent. of cases in the course of six months or a year.

The chief precaution to be observed is to teach the patient how to support the dilated ring with the finger during exercise (Fig. 5), and if it seems inadvisable to trust this to his intelligence, a truss should be worn throughout the treatment.

I would ask your attention to the following exercises which have proved to be of value in overcoming the premonitory symptoms already described and in curing the actual hernia even in severe cases.

Before beginning the exercises, of course the hernia should be reduced and retained, if it is a recent case with pain, by a truss, especially if the hernia was due to sudden strain, and rest should be taken until any inflammation has disappeared and the truss can be worn with comfort.

The greatest stress should be laid on teaching control of

FIG. 6.



Position described in Exercise 4, showing the correct position of the legs and of the right hand.

FIG. 7.



Left side lying, showing right hand protecting the right abdominal rings, the surgeon assisting in trunk flexion to the right.

FIG. 8.



Powerful contraction of the right external oblique described in Exercise S. Rings on the right side protected.



the abdominal muscles by forced breathing, abdominal and thoracic, and for this purpose the first exercise should be as follows:

*Exercise 1.*—Patient lying on back; place one hand across abdomen, the other protecting the ring; inhale deeply; exhale by pressing on the abdominal wall until voluntary contraction has been acquired, when this movement can be done without placing the hands over the abdomen. (Fig. 5.)

*Exercise 2.*—Patient lying on back, one hand across abdomen, the other protecting the ring. Inhale and exhale without drawing in the abdomen. In this way control of the abdominal wall is obtained, while the hernia is protected by placing the finger over the external ring. In most patients it is possible to teach them in one or two sances how to find the external ring and how to protect it in the various exercises.

*Exercise 3.*—Patient lying on the back, one hand behind the neck, the other covering the external ring. Raise the head and shoulders, twisting in the opposite direction from the hernia. In this way the oblique muscles of the affected side are put into strong contraction, but if the movement be symmetrical the rectus alone will receive the strain. (Fig. 4.)

*Exercise 4.*—Patient lying on back, external ring protected. Without bending the knees raise both feet six inches from the table. Alternate the raising and lowering of the feet, twelve inches, five times without touching the table. (Fig. 6.)

*Exercise 5.*—Patient lying on the back, ring protected. Raise body from the lying to the sitting position, with the shoulders twisted so that the shoulder of the affected side is forward.

*Exercise 6.*—Left side lying, for hernia on right side; left neck firm; right hand protecting the ring; side flexion of trunk. (Fig. 7.)

*Exercise 7.*—Patient standing, one hand protecting the ring, the other behind the head. Circumduction of the trunk, with side bending from the affected side.

*Exercise 8.*—Patient seated; hips firm; backward bending and twisting shoulders to the left in a right hernia. (Fig. 8.)

Each exercise should be repeated twenty times.

*Exercise 9.*—Massage, consisting of circular kneading movement, beginning at the external abdominal ring and passing upward and outward to the anterior superior spine.

The general regulation of the physical life of these patients is also of importance. Perhaps the greatest mistake is to forbid any active exercise. I find that there are few forms of exercise that such patients may not indulge in, not only with safety but with great benefit. It may be laid down as a safe rule that the two things in exercise to be avoided are sudden and severe strain requiring the breath to be held, and, secondly, the maintenance of standing positions for long periods.

I believe that one can engage in such games as tennis, golf, or even bowling, cricket, gunning, dancing, swimming, canoeing, and most forms of gymnastics, without danger if reasonable precautions as to support are used; but one should avoid football, hammer-throwing, jumping, riding, shot-putting, and wrestling. This last sport might be considered as the most dangerous exercise that could be taken by such patients.

The exercise, *par excellence*, recommended by Dr. Lucas Championniere, in his admirable work on hernia, in which he also takes up the treatment by trusses and by operation, is bicycling, and he lays great stress on the fact that the weight is not supported by the legs, and that there is no possibility of violent and unsuspected strain, and that the dose can be accurately indicated.

The object of the exercise treatment for acquired inguinal hernia is twofold:

1. To strengthen and increase the number of muscle fibres in the transversalis and two oblique abdominal muscles, and so to reinforce the aponeuroses with which they are connected.

2. To cultivate alertness, control, and self-consciousness in these muscles, thus causing them to respond instantly and automatically to any sudden strain that may be thrown upon them. More harm may come from surprising a strong muscle which is relaxed and out of control than can come from the same strain on a weaker muscle that is ready for it.

One must not lose sight of the general effect of these exercises on stimulating the intestines and regulating the action of the bowels.

I will close with a brief review of four cases that may be called typical of the conditions I have been trying to describe.

Mr. G. acquired a complete right inguinal hernia at 5, coming on after a long and exhausting walk. This remained down for several years, but he has been entirely free from it ever since. The external rings on both sides are still larger than normal, admitting two fingers, but his muscular system is well developed and he is able to go into the heaviest gymnastics without symptoms.

Mr. K., the second case, at the age of 12, shortly after climbing and swinging in a tree, noticed a visible protrusion, which has remained in the same condition ever since.

For five years he wore a truss, which had no effect, and the hernia has remained much in its original condition up to the present time. Walking makes it painful, but he has taken part in games like base-ball and canoeing without inconvenience. This is a case in which the prognosis would be good for a cure in six months or a year.

The third case, L. M., was referred to me three years ago by Dr. Robert G. Le Conte. The patient was 54 years of age, had a double hernia for ten years, but did not wish to undergo operation. He came to me wearing a truss, and after practising these exercises for six months the sense of insecurity left him and he dispensed with the truss, except during violent exercise. After three years, his abdominal wall is in excellent condition, with no signs of return.

The fourth and last case, J. M. W., age 50, also referred to me by Dr. Le Conte, March, 1911, complained of severe pains of a dragging character in the right inguinal region of  $3\frac{1}{2}$  years' duration, during which time he wore a truss. He had to give up riding, walking, and golf, and confined himself to office work.

An examination showed patulous abdominal walls and distended right external ring, with a distinct impulse on coughing. There was, however, no actual hernia.

A course of three months with those exercises completely cured these symptoms and gave him a sense of security which has enabled him to take part in the many activities from which he has hitherto felt himself debarred. He has had no return of his symptoms.

The prognosis for cure in these cases is about 70 per cent., according to Seaver, but I have not had a sufficient number of cases long enough under my own care from which to draw conclusions as to the percentage. It does, however, seem to be conclusively proved that there is a wide field for the non-operative treatment of inguinal hernia, besides the usual makeshift of applying a truss which may fit imperfectly and which frequently causes more inconvenience than the hernia itself.

## A YEAR'S WORK IN APPENDICITIS

BY JOHN B. DEAVER, M.D., L.L.D.

PHILADELPHIA

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It is an encouraging sign of the times when the number of operations for chronic appendicitis exceeds those of the acute disease. It is also far better for the patients, as I shall show.

This paper covers the work done at the German Hospital and the Children's Hospital of the Mary J. Drexel Home, as well as that of my clinic at the University Hospital. During the past year 561 chronically diseased appendices have been removed without a death. In the same time there have been 356 cases of acute appendicitis with 8 deaths.

That the existence of chronic appendicitis is productive of symptoms no longer admits of question. The question of real importance is the diagnosis of the condition. Usually it is anything but difficult, necessitating only the patient's brief recountal of symptoms, supplemented by palpation of the right iliac fossa; still, often enough it may be a matter of the greatest difficulty.

This is due to the facts that, first, chronic appendicitis does not always give symptoms referable to the right iliac fossa, and, secondly, that symptoms referable to the right iliac fossa are not always due to a diseased appendix. The train of symptoms set up by a diseased appendix may be referable to a distant point, most commonly the epigastrium. Stomach trouble often turns out to be appendix trouble. Pain in the epigastrium, distress after eating, nausea, belching, vomiting, the protean forms of indigestion may be caused by chronic appendicitis without symptoms of palpable evidence of disease in the right iliac fossa. Certain of these cases, if watched closely, will have slight exacerbations of the local process during which the diagnosis may become clear. In other cases the state of affairs will become evident only by abdominal section, when a negative examination of the upper abdominal viscera, coupled with the finding and removal of a chronically diseased appendix, followed

by recovery from symptoms, will substantiate the diagnosis. Many other cases will present insufficient symptoms to warrant exploratory incision, and will drift along to spontaneous recovery or to confirmed dyspepsia with involvement of the other organs of digestion.

I know of no way to diagnosticate chronic appendicitis without pain and local symptoms. Pain in some form or degree is our greatest reliance, yet it has become evident to me that chronic appendicitis is not always accompanied by pain. Since I have been removing many appendices as an incidental procedure when operating in the neighborhood it has been found by the pathologist that a large percentage of these appendices present unmistakable evidence of past or present inflammation. It would seem that local pain does not necessarily accompany inflammation, but is present only when colic is caused, when the intra-appendicular tension is raised by stricture or retention, or when the serous surface is involved in the inflammation. This is not surprising, in view of the observation of Lennander and others showing that the abdominal viscera themselves are quite insensitive to pain. In a few cases chronic appendicitis proceeds by slow stages to complete obliteration of the organ, which is Nature's method of spontaneous cure, without noteworthy symptoms of any kind and without any symptoms of the sort that we now recognize as pointing to the appendix. These remarks will illustrate the difficulty of recognizing existing chronic appendicitis, though it may be present in an advanced stage, and, if space permitted, I could cite many cases in point.

The second difficulty relates to symptoms simulating disease of the appendix. These may be due to movable right kidney, to renal or ureteral calculus, to cholecystitis, pancreatitis or duodenal ulcer, to right-sided disease of the ovary or fallopian tube, to bands or adhesions such as are commonly spoken of as Lane's kink of the ileum and membranous pericolicitis, to an abnormally movable cæcum, and, finally, to one of a number of very uncommon conditions, such as typhlitis, suppuration at the internal ring, etc.

I am led to speak of these conditions because of the fact that the operation of appendectomy for symptoms believed to be due to chronic appendicitis, though almost uniformly successful in bringing relief, does not in every instance do so. When symptoms persist after the operation they may be due to several causes. First,



adhesions may be present either as a result of the appendicular disease, or the operation itself; secondly, secondary changes in other organs may have occurred—for instance, there is reason to believe that cholecystitis and cholelithiasis and ulceration of the stomach or duodenum may depend in some obscure way upon chronic appendicitis; third, the functional derangement, commented upon previously, when of long duration may have become engrafted as a habit upon the physiological activities of the alimentary tract; fourth, the diagnosis may have been mistaken.

The chief safeguard against mistaken diagnosis is a careful history and the following up by more extended examination of the leads that may be obtained.

Movable or diseased kidney, renal or ureteral calculus, upon careful inquiry, will usually show in the history some abnormal character of the urine, of the act of micturition, or a characteristic type of pain. The floating kidney can be palpated and the presence of stone may usually be confirmed by the X-ray and cystoscopy.

It must not be overlooked, however, that chronic appendicitis may occasionally give pain referred to the genitalia or the leg. Cholelithiasis, cholecystitis, chronic pancreatitis, and duodenal ulcer, when they give rise to the typical symptoms, are in no danger of being mistaken for chronic appendicitis, but we have long since learned that they may give indifferent symptoms with no pathognomonic value, simple indeterminate dyspepsias, and when this is true they are in danger of being mistaken for the less outspoken varieties of chronic disease of the appendix. Only those who never follow their diagnoses inside of the abdomen can harbor the delusion that these differentiations are always possible.

Disease of the right fallopian tube or ovary will also, as a rule, give symptoms that are definitely related to the sexual history and function, and the condition may be demonstrated, though not invariably so, by examination. Usually the signs and symptoms are bilateral, tenderness is lower in the pelvis, nausea, vomiting, and digestive derangements are less frequent, and the regularity of exacerbations at the menstrual period is very significant. Here we must mention that chronic appendicitis may give pelvic symptoms in the absence of pelvic disease, and that the two conditions may co-exist. With a history of pelvic disease and a negative examina-

tion of the internal genitalia the possibility of chronic appendicitis should always be strongly considered.

The attention which has recently been focused upon the presence of abdominal bands and membranes in the region of the cæcum has resulted in demonstrating that these formations are not infrequent and that they may upon occasion give rise to symptoms which are very like those of chronic appendicitis. The same is true of an abnormally movable cæcum, which has been emphasized by the Germans, and recently by Sailer in Philadelphia. Commonly the obstructive element and intestinal stasis, with its train of symptoms, are more likely to be prominent in these conditions. There is no way of differentiating with certainty before operation and inspection. Much is claimed for the X-ray in the diagnosis of these conditions, and doubtless there is a percentage of cases in which help can be obtained. Skiagrams of abdominal viscera are, however, apt to be misleading, and particularly so the farther away they are from the orifice through which is introduced the bismuth or other mixture that is needed to outline the viscus. Too much importance should not, therefore, be attached to skiagraphic diagnosis.

It remains for the surgeon to satisfy himself at the time of operation that the appendix is such as to account for the symptoms. This he should do by inspection or palpation before concluding the operation.

I am not greatly impressed with the cry that has at times been raised as to the unsatisfactory results of appendectomy in many cases of supposed chronic appendicitis. To me it has seemed that no operation is more uniformly satisfactory to the patient from the stand-point of relief of symptoms. The elimination of unsatisfactory results depends more upon the care with which the diagnosis is made than upon any other factor, and since we have recognized that many atypical symptoms may be due to chronic appendicitis, and are operating for their relief, it is well to utter a word of caution that in the cases which give no history of exacerbations that can be easily recognized as appendicitis especial care must be used in assuring ourselves that we are dealing with appendicitis and not with one of the conditions that may simulate it.

In acute appendicitis the situation is the reverse of that in chronic appendicitis in respect to diagnosis and treatment. The acute forms

of the malady are, as a rule, easily recognized by one of experience in abdominal disorders, while the treatment is difficult, and methods vary widely in skilled hands, and not a few important questions yet await settlement.

Perhaps the most important question that concerns the internist and practitioner, as well as the surgeon, is the use of purgatives during the acute stage of the disease. It is not long since we all thought that a purge should be administered in the initial stages of the disease, but we have come to realize that the contents of the bowel play practically no part in the continuation of the disease, while the peristalsis induced by purgation has proved to be a most unfavorable factor, defeating the end and principle upon which our treatment is based, namely, anatomic and physiologic rest. The purgative is more deadly than the scalpel. Of this I have long been convinced, but, with a view to obtaining exact figures to support my contention, I have had the year's work analyzed with respect to this point. Among 259 adults there were 79 that had a history of being purged before admission. Of these 79 cases all save 2, at the time of operation, showed appendices that were either perforated, gangrenous, acutely ulcerated, or surrounded by an abscess. The remaining two only were simple catarrhal inflammations. Of the 7 deaths occurring in the series 5 were purged before admission. Of those drastically purged 97.5 per cent. showed the severest kinds of appendicular disease. The exact figures are here given:

Pathologic lesion of appendix: Perforation, 30; gangrene, 9; acute ulcerative and suppurative, 23; peri-appendicular abscess, 15; acute catarrhal, 2.

Among 100 children the same rule holds. There were 56 that had been vigorously purged. Of these 86 per cent. showed the more serious lesions, such as gangrene, perforation, abscess, etc. Twelve cases had diffuse peritonitis, all of which had been purged before admission. The table is as follows:

Pathologic lesion: Perforation, 22; gangrene, 15; acute ulcerative, 6; peri-appendicular abscess, 4; acute catarrhal, 8; indefinite, 1.

In this series there were 44 acute cases that were not purged, and in this group there was but one case of perforation, 8 cases of gangrene, and no peri-appendicular abscesses. The remainder showed

acute catarrhal or ulcerative inflammations which were strictly confined to the appendix. Consider these figures and tell me whether the mortality of appendicitis is medical or surgical. The time is not far distant when the man who uses a purge in acute appendicitis will lose his standing in the profession as well as his patient. In these cases the enema should always be used when it is desired to move the bowels, but in general less attention should be paid to evacuation of the bowels and more attention to placing them at rest and to the abatement of peristalsis.

Especial care is necessary in the treatment of appendicitis in children. It is by no means uncommon, but is frequently overlooked on account of the prevalence of colics and gastro-intestinal derangements among the young. Unquestionably fermentation and gastro-enteritis often are best treated by purgation, but before beginning this treatment appendicitis should be carefully excluded. In general, the disease in children is identical in its pathology and clinical manifestations with that seen in adults. We do find, however, that the pathology not infrequently moves with startling rapidity; gangrene, perforation, and diffuse peritonitis, all within the course of 24 hours or less, not being exceptional. On the other hand, children resist continued sepsis more vigorously than adults. From the operative stand-point we find that children are more subject to shock, but unless this ends fatally their recuperative power rapidly asserts itself.

The medical treatment of acute appendicitis, in my opinion, consists of the following:

1. Diagnosis.
2. Avoidance of purgative or laxative medicine.
3. The institution of anatomic and physiologic rest.
4. Immediate preparation for removal of the appendix.

Anatomic and physiologic rest are secured by the following measures:

1. Complete rest, preferably in the sitting posture to aid in the gravitation of exudates, if any, toward the pelvis.
2. Absolute prohibition of anything by mouth, including water and ice, as well as nourishment, in order to quiet peristalsis.



3. Gastric lavage to relieve the stomach of accumulated fluids, preventing, in large degree, toxæmia, vomiting, and peristalsis.
4. The constant instillation of normal salt solution by way of the rectum according to the method devised by Murphy. This diminishes thirst and supplies the need of water.
5. Ice-bags over the abdomen to quiet pain, to assist in keeping the patient quiet, and, more particularly, to discourage meddling examinations.

I have said nothing about drugs, for they are not necessary. Usually they are a disadvantage. Gastric sedatives are worse than useless. Morphine, if given indiscriminately, masks the symptoms, interferes with the phagocytic activity of the leucocytes, and inspires both patient and physician with false confidence. The pain of appendicitis is not often so great as to require morphine, but if necessary it should only be given in a minimum dose after decision for operation has been made. Under the above treatment the pain abates and no anodyne is needed. In the German Hospital and the Children's Hospital of the Mary J. Drexel Home it is never found necessary to use opiates prior to operation, and I cannot believe that the disease is so different elsewhere. It is far better never to use them than to use them once too often. In the rare cases where operation is impossible they may, of course, be given in accordance with the indications of extreme restlessness and pain.

This series bears out what we already had established regarding the liability of recurrence of attacks, for of the 359 cases, 170, or practically one-half, had had previous attacks. This means that 170 individuals, either of their own volition, or more commonly upon the advice of their physician, in order to avoid operation had bartered the operative mortality of uncomplicated cases, in this series nothing and never great, only to be obliged to accept later the risk of the general mortality of this series—4.2 per cent. Three of these patients, all of whom had been drastically purged, died unoperated, being admitted in a state of advanced diffuse peritonitis that precluded either operation or improvement. This leaves an operative mortality in all acute cases of 2.7 per cent. in adults and 1 per cent. in children. Nor is this the only difference, since recovery in uncomplicated cases was almost without exception speedy



and without complication, while many who recovered after acute attacks did so only after a battle with sepsis, which depleted their physical powers, wasted their resources, and, at times, left impairments, such as fecal fistula, phlebitis, adhesions, and ventral hernia, while one case developed postoperative insanity.

In females the obligation to advise early operation is pressing because of the danger of tubal involvement. In 7 cases I found the appendix tied up in an inflammatory mass with the right tube and ovary, and in 17 tubo-ovarian disease co-existed.

As a factor in sterility appendicitis possesses considerable importance and is not an infrequent cause of pelvic inflammation in young females. From the stand-point of mortality, however, the female sex possesses an advantage, since they resist peritoneal, and especially pelvic, inflammation better than the male. Of the 7 post-operative deaths among adults all were males.

A factor over which we have no control, but which greatly influences the prognosis, is the situation of the perforations. Those which occur at the base rapidly give rise to a peculiarly virulent type of peritonitis and are likely to cause death. The simple reason is to be found in the fact that a hole in this location allows the teeming bacterial life of the cæcum to pour out into the abdominal cavity in overwhelming dosage. Another highly dangerous form of the disease is that in which the appendix, lying behind the cæcum and ascending colon, causes retrocecal suppuration, and infection of the cellular retroperitoneal tissues, where drainage is difficult and absorption rapid. Infections in the central region or enteronic area among the coils of small intestine are also difficult to handle and exceedingly likely to cause diffuse peritonitis. The most favorable areas from the stand-point of treatment are the marginal or lateral and the pelvic zones. These locations are influenced chiefly by the location of the appendix, which we are unable to influence or determine beforehand. Consequently our only resource is prevention by removal of the appendix at the outset before it can induce suppuration of any area.

In regard to the treatment of peri-appendicular abscess I am still a believer in the method of extraperitoneal approach when it is feasible. Many abscesses have, as a part of their retaining wall, the parietal peritoneum of the right iliac fossa or the loin. When

Nature has thus excluded the infectious area from the general cavity. I would not think of attacking the collection transperitoneally by tearing down the defensive adhesions between the intestines and omentum. Much simpler and much safer, in my opinion, based on results, it is to open directly into the abscess through the abdominal wall. There are many cases in which the abscess is not in so favorable a situation and must be attacked transperitoneally. In this case protection to the general cavity must be secured as well as may be by the disposal of gauze packs during operation and the arrangement of drainage when the infected area has been dealt with. Even here I do not break up adhesions unnecessarily, but only enough to deal thoroughly with the abscess cavity. The proposal of some surgeons to relieve all the adhesions is contrary to reason and results. It is not the breaking up of adhesions, but the institution of drainage that cures. It is a general rule that we cure best when we work with Nature rather than by flouting her efforts and substituting our own works for those of our Creator.

As a corollary to this attitude regarding peri-appendicular abscess I take issue with those surgeons who never leave the appendix.

In the 356 operated cases of acute appendicitis upon which this paper is based the appendix was not removed in 15 cases, 4.2 per cent., and I have not yet had cause to regret leaving it in any of these cases. True, there is a slight danger of recurrent attacks in such a case, but this can be obviated by a second operation for removal of the appendix at the same time that the operation for the inevitable ventral hernia is performed. The appendix need not be left in any case that can be so securely closed as to obviate incisional hernia, so that a second operation is not often avoided by those who insist upon its removal. To remove it certainly does not assist in recovery from the attack itself, while the breaking up of adhesions and burrowing about in the inflammatory exudate in the effort to locate a buried appendix are certainly dangerous. Even were it possible, by removal of the appendix, to secure better closure of the abdominal wall, I would still not do it in certain cases, for it has always been my belief, frequently stated, that a living patient with an appendix is better than a dead patient without an appendix. I always make an effort to find the appendix, and usually am successful without the

infliction of much damage, but there is a limit beyond which I will not go even in the interest of thoroughness, much preferring the interest of the patient.

Another subject for serious disagreement among surgeons relates to the time of operation in cases of spreading peritonitis. There can be no disagreement as to the advisability of operating early. When the disease is still confined to the appendix, even in acute appendicitis, the technique is as simple and the results just as satisfactory as in chronic disease. The difficulty and the danger arise when the inflammation has passed beyond the confines of the appendix, whether by perforation or by extension through its walls without gross perforation. Whatever the cause of the initial lesion of the appendix, the peritonitis resulting from it does not present an extraordinarily diverse flora. Thus in the series under consideration in the cases of peritonitis we found the following organisms: *Bacillus coli*, 171; *bacillus pyocyaneus*, 7; *bacillus fecalis alkaligenes*, 5; *Staphylococcus albus*, 5; *Streptococcus*, 2.

The preponderance of colon bacillus is natural in infections derived from this region of the alimentary tract, and gives considerable uniformity to the picture of appendicular peritonitis. *Bacillus pyocyaneus* is somewhat more dangerous than the colon bacillus. *Bacillus fecalis alkaligenes* runs practically the same course as the other members of the colon group.

The *Staphylococcus albus*, which has been so loudly proclaimed by Dudgeon and Sargent as the friend of the peritoneum, "the first to come and the last to go," we do not find so uniformly, though we have made many cultures in exactly the manner prescribed by these authors. We can confirm their results, however, in respect to its relative harmlessness.

The *Streptococcus* of appendicular inflammation does not appear to possess the same pathogenicity as is usually associated with the *Streptococcus* elsewhere. This may be due to the fact that the *Streptococcus fecalis* is different biologically from the *Streptococci* found in the sputum, in erysipelas, or in uterine and cellular infections. It reacts differently toward the test sugars, and may well do so in the human body. Or the difference may be due to the location, the better opportunity for drainage, or to some other clinical reason.

At any rate, streptococcic peritonitis of appendicular origin is not so fatal as streptococcic peritonitis secondary to other intra-abdominal lesions, though it is more serious than colon infections.

Now, when these organisms reach the peritoneal cavity, the result depends upon three factors: (1) the virulence of the organism, (2) the resistance of the body, local and general, (3) the dosage or amount of infection. Over the first two factors we have little control. Our influence is to be directed chiefly upon the last factor. By early operation we can rescue the peritoneum before the infection becomes too great to counteract. Just as our laboratory animal will react from .5 C.c. of a virulent culture of colon bacilli when 5 C.c. would cause a fatal peritonitis, so will our patient recover from early peritonitis and a limited infection when an additional amount will give rise to a diffuse peritonitis. In this early stage of diffusing peritonitis, which is still local though unconfined, we should operate at once. Clinically, this stage extends well up towards 40 hours after the onset of the disease, though there are cases in which the process is more rapid than this. If we remove the appendix, prevent it from pouring out more infective material into the peritoneal cavity, the dosage of bacteria is not yet sufficiently great to set up a diffusing peritonitis, but is speedily overcome by the resisting mechanism of the body.

If we fail to operate in this early stage, peritonitis results, and, if so rapid and virulent that it marches ahead of the formation of plastic peritoneal adhesions, which seek to confine it to its origin, we have a diffuse peritonitis. This peritonitis, moreover, may be no more dependent upon the presence of the infecting focus—in this case the appendix—than are the flames which devour a house dependent upon the match which set it afire. The disease is peritonitis and not appendicitis. The fireman must direct his hose against the house and not turn his chemical extinguisher against the match. Can we treat diffuse peritonitis by operation? From an experience covering the whole development of the modern treatment of peritonitis, I say, no. I have washed, wiped, drained with gauze, and with tubes, employed antiseptics, and run the gamut of active procedures proposed for treatment, I can only echo the words of an English surgeon that “the more thorough the operation the more



quickly the patient dies." After reducing the treatment to simpler and simpler lines, with corresponding improvement in mortality as each successive procedure was abandoned, I have come to the conclusion that no operation should be done in the presence of diffuse peritonitis, and have in this way achieved better results than by any form of operation, however complicated, however simple. Remember, that these remarks do not apply to local peritonitis, whether confined or unconfined. They apply only to those cases, usually of more than 40 hours' duration, in which the clinical evidence of widespread peritonitis is present. Such cases appear generally sick, the face flushed, respiration chiefly costal in type, the temperature elevated to 102° F., though it may be normal. The abdomen may be beginning to distend, the tenderness is general, though usually most marked in the region of the appendix. Peristalsis is brief and tinkling, constipation absolute or, at least, obstinate. Such a patient is not a subject for operation. We cannot drain the entire abdominal cavity, and removing the appendix, though that were desirable could we "wish" it out, does not aid the body to overcome the infection in the outlying areas where the conflict is being waged that determines the life or death of the patient.

Operation, with its depression of the vital powers and resistance, will tip the scale against the patient. Masterly inactivity, with appropriate treatment, will tide them over the crisis, allow victory in the remote areas of the peritoneum, and localize the infection in the neighborhood of the appendix, where it may be dealt with to greater advantage and with every prospect of success. The treatment is the same as that already spoken of as anatomic and physiologic rest. If we treat our patients in accordance with these principles, it is true we shall still have a mortality in dealing with this grave disease, but it will be chiefly the mortality of weakness, of cardiac, pulmonary, or renal complications, and not that of peritonitis, our greatest foe. In this series I lost three cases from peritonitis, which I fear were due to inability to apply correctly these principles. The remainder died of cardiac and pulmonary complications.

In conclusion I would cite the collected data upon the cases of acute appendicitis.



## ACUTE APPENDICITIS

## Records of The German Hospital, 1911

Number of cases operated.....	256	
recovered .....	249	
died .....	7	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 3em; vertical-align: middle;">}</div> <div>Acute dilatation heart, 1. Pneumonia, 3. Peritonitis, 3.</div> </div>
Number of cases of death without operation....	3	
Number refusing operation.....	15	
Percentage of deaths.....	2.7	
Females .....	102, or 40 per cent.	
Males .....	154, or 60 per cent.	
Of the 7 postoperative deaths all were males.		
Number of cases of peritonitis..	140, or 54.68 per cent.	
Number of cases having repeated attacks .....	108,	
Number having localized peritonitis .....	60	} On admission.
Number with diffuse peritonitis .....	41	
Appendices removed .....	245, or 95 per cent.	
Appendix not removed .....	11, or 4.3 per cent.	
Number of cases drained ....	118, or 45.6 per cent.	
Number of cases giving a history of being purged before admission.....	79	

## PATHOLOGICAL LESION.—

Perforated or self-amputated, etc. ....	30
Gangrenous .....	9
Abscess .....	15
Acute ulcerative, exudate, etc. ....	23
Acute catarrhal .....	2

Cultures were taken on 179 cases, and the following organisms isolated:

Bacillus coli .....	120
No growth .....	44
B. pyocyaneus .....	6
B. fecalis alk. ....	4
Staph. albus .....	3
Streptococcus .....	2

## COMPLICATIONS.—

Broken-down wounds .....	37
Superficial .....	17
Deep .....	20
Of these 4 were with salpingitis, and one with tuberculous lymphangitis.	
Fecal fistula .....	14
Of which 9 were purged before admission.	
Ulcerated cæcum .....	4
Bronchitis .....	4

Pneumonia .....	4
Subdiaphragmatic abscess .....	6
Secondary collection .....	3
Tubo-ovarian disease .....	17

Of these 7 were with the appendix tied in the mass of the right ovary, etc.

Congestion of the lung, bronchopneumonia (?) .....	2
Intestinal obstruction .....	2
Pleural effusion .....	1
Acute cardiac dilatation .....	1
Phlebitis .....	1
Urinary fistula .....	1
Epididymitis .....	1
Postoperative insanity .....	1
Lane's kink .....	1

## ACUTE APPENDICITIS

Records of The Children's Hospital, Mary J. Drexel Home, 1911.

Number of cases operated.....	100	
recovered .....	99	
died .....	1	
Number of cases refusing operation .....	3	
Percentage of deaths .....	1	
Females .....	39, or 39 per cent.	
Males .....	60, or 60 per cent.	
Number of cases having previous attacks .....	62	
Number of cases distinctly localized to appendix (not peritonitis) .....	12	
(Local-izing) { Number of cases having cloudy fluid....	17	} On admission.
{ Number of cases having diffuse peri-		
tonitis .....	32	
Number of cases having localized abscess (mass) .....	24	
Number of cases subsiding (not peritonitis) ..	8	
Number of cases subsided (not peritonitis)...	7	
Number of cases with appendix removed....	96, or 96 per cent.	
Number of cases appendix not removed.....	4, or 4 per cent.	
Number of cases drained .....	53, or 53 per cent.	

## CONDITION OF APPENDIX IN CASES PURGED

Number of cases giving history of being purged before admission..... 56  
 86 per cent. of the 56 cases purged were of the gangrenous, etc., nature.  
 All but 8 cases being either gangrenous, perforated, etc., these being classed as acute.

Acute inflammation confined to appendix.....	8
Gangrenous .....	15
Perforated .....	22
Abscess .....	4

Acute, ulcerated, exudate, etc. ....	6
Indefinite .....	1

12 of these cases had diffuse peritonitis—classified under gangrene or perforated appendicitis, all of whom were purged.

#### CONDITION OF APPENDIX IN CASES NOT PURGED

Of these there were 43, 8 of which were gangrenous, 1 of which, or 14 per cent., was perforated.

The rest showed ulceration of mucosa, or acute catarrhal inflammation.

Cultures were taken in 76 cases and the following organisms isolated:

B. coli .....	51
Staph. albus .....	2
No growth .....	21
B. pyocyaneus .....	1
B. fecalis alk. ....	1

#### COMPLICATIONS.—

Abscess between layers of mesentery .....	2
Mesenteric lymph glands enlarged .....	1
Secondary collection, glands of mesentery .....	1
Fecal fistula .....	5 (All were purged.)
Ulceration cæcum .....	1
Influenza .....	1
Nephritis .....	1
Lane's kink .....	1
Postoperative pneumonia .....	3
Multiple localized abscesses at operation .....	3
Pelvic phlebitis .....	1
Suppurating mesenteric glands .....	1
Toxic diarrhœa .....	1
Broken-down wound .....	1
Twisted ovarian cyst, gangrenous, with appendix adherent to it .....	1
Bronchitis .....	1
Subdiaphragmatic abscess .....	1

# Ophthalmology

## NOTES ON SOME INTERESTING EYE CASES

BY LESLIE BUCHANAN, M.D.

Surgeon to the Glasgow Eye Infirmary

MUCH interest has been excited amongst ophthalmic surgeons within the last few years by the advance in the operative treatment of chronic glaucoma, and one feels that any unusual facts in connection with glaucoma should be put on record, as they may assist some one in the future.

The four cases which I bring forward here appear to me to be of sufficient interest to merit description, two showing good results under the old, and two under the new method of treatment.

I shall recite the cases as briefly as is consistent with clearness, but, since each was under observation for a long time, it is not advisable to be too brief.

### ACUTE GLAUCOMA IN BOTH EYES

CASE I.—M. S., æt. 55, came to the Eye Infirmary under my care August 1, 1902, complaining of dim vision of right eye. This came on suddenly, with pain in the head and sickness, three weeks previously.

V. A. Rr. fingers at 10 ft. V. A. L.  $\frac{20}{10}$ .

Right eye: Cornea hazy; pupil dilated and fixed. Tension + 2. O. E. R.: Fundus dimly seen. Nerve deeply cupped and very red. Left eye practically normal. Tension normal. O. E. L.: Fundus clear; nerve not cupped but distinctly congested. H + 2. Gtt. eserine prescribed. Right pupil does not contract well; admitted as an in-patient.

August 9, 1902. Right eye. V. A. fingers at 6 inches only. T. + 2; pain still severe.

August 10, 1902. Large wide peripheral iridectomy made under cocaine.

August 21, 1902. Eye quiet and well healed; patient dismissed.

August 29, 1902. Rt. T. N. V. A.  $\frac{20}{10}$ , unaided.

October 17, 1902. Rt. T. N. Media clear. V. A.  $\frac{20}{30}$ .

January 27, 1903. Right eye, T. N. Media clear; nerve cupped, not deeply, but widely. V. A. unaided  $\frac{20}{30}$  and aided  $\frac{20}{15}$ .

The patient was seen at intervals for a few months longer and then was lost sight of.

She was next seen on December 3, 1906, complaining of great pain in the left eye of a week's duration. She had been washing clothes and "thought it was cold or would have come sooner."

The left eye was in a state of acute glaucoma, having all the usual appearances, with tension = + 2 or more. V. A. L. = fingers at 1 foot only. Right eye in same state as at end of January, 1903. To cut the story short, the left eye was operated upon and, after two months, the vision was sufficiently good to enable her to get glasses with which she saw  $\frac{20}{18}$  Rt. and  $\frac{20}{25}$  L. Later the left eye improved still more and aided vision was found to be  $\frac{20}{15}$ , left.

This is the only patient I have seen and operated on on account of acute glaucoma in both eyes.

#### CHRONIC GLAUCOMA

The next case is one of chronic glaucoma in a myopic subject, in which iridectomy in the worse eye was followed by prompt and permanent relief, whereas, in the better eye, the operation had to be repeated.

CASE II.—T. P., æt. 47, had suffered since 1896 from dim vision in the left eye. This came on somewhat suddenly and had remained stationary until August, 1902, when I first saw him. There was a constant haze over the left eye, and only a year ago (1901) a temporary haze over the right eye was first noticed. The haze in the right eye appeared at irregular intervals, becoming more frequent lately. In August, 1902, V. A. R.  $\frac{15}{65}$ , and aided  $\frac{15}{20}$ . V. A. L.  $\frac{15}{65}$ , and aided  $\frac{15}{25}$ . Ophthalmoscopic examination of both eyes shows cupping of the optic nerves, that of the left being more advanced than that of the right. Tension Rt. +. L. + 1. Visual fields contracted concentrically. Eserine ordered for both eyes.

For a month matters remained stationary, the haze coming and going, but sometimes lasting as long as six hours, and operation was strongly urged upon the patient. The haze had been very intense for a whole day on the left eye and slight on the right also, so operation was performed September 27, 1902.

A large, very wide and peripheral iridectomy was made upwards in the left eye. It was noted as a striking fact that when the sclera was perforated, a large amount of venous blood escaped and continued to flow for some minutes. The only event of note in the healing was a marked and persistent œdema of the conjunctiva.

The result of the iridectomy in this (the worse) eye was good at once. Vision rapidly rose, the haze cleared away and has never returned in any shape or form during more than nine years. On November 8, 1902, V. A. R.  $\frac{14}{65}$  and  $\frac{14}{20}$  aided. L.  $\frac{14}{100}$  and  $\frac{14}{16}$  aided. The left is now the better eye.

November 9, 1902, an iridectomy was made up in the right eye. As this eye had not been so bad as the other, the coloboma was made of medium size, but very peripheral. Bleeding of the same venous character as took place in the case of the left eye occurred here, but no œdema of the conjunctiva followed. Healing was uneventful and all went well for a month. Aided vision had been



raised by correction of astigmatism resulting from the corneal wounds to  $15/13$  each eye.

Six weeks after the operation in the right eye a slight haze was noticed before it. This recurred at intervals and I became convinced that a larger iridectomy was required. Ultimately the recurrences of the haze in the right eye became more marked, and on February 29, 1903, I again operated on the right eye, drawing out the pillars of the iris and enlarging the coloboma to fully the size of that in the left eye. The eye healed quietly and well, and the result has been that, with one or two trivial recurrences, the disease terminated and the patient has had no further trouble. Aided vision when last tested (1905) was  $15/14$  each eye. When last seen (1911), both eyes were still well, no haze being present.

#### CHRONIC GLAUCOMA OR SEROUS CYCLITIS: TREPHINING

The next case was one not easily named. The symptoms were those of chronic glaucoma, but the age of the patient made serous cyclitis a more probable diagnosis.

CASE III.—A. T. L., æt. 34, complaining of attacks of obscuration of vision of the right eye, was first seen at the end of 1908. The history was that dimness of vision first became noticeable about the end of 1904. It was treated with atropine and iodide of potassium for over a year, but without benefit. There were spots on the posterior surface of the cornea and the tension of the globe was elevated. The right eye remained as it was under this treatment till the end of 1907, when the treatment was discontinued and no other taken up. The eye gradually got worse, and when I saw him in October, 1908, V. A. R. =  $15/80$  unaided but correction of myopic astigmatism  $15/15$  dimly. L.  $15/20$  and corrected  $15/12$ . Some spots of keratitis punctata were still present in right eye, more in left. Tension, right +. L. normal. Ophthalmic examination: Deep and wide cupping of optic nerve of right; left, normal. Eserine was ordered and, as there was a slight history of tuberculous bone lesions, Syr. ferric iodide was also prescribed. The condition of the eye remained very much the same, often growing worse at night and better in the mornings. During one month there were sixteen bad days, *i.e.*, days when the vision became dim, and fourteen good days, when there was no dimness. Eserine and dionin ordered.

On April 22, 1909, the eye grew very much worse, the vision being reduced to perception of light only. The tension was then + 2, the cornea steamy and the ocular conjunctiva congested. Local applications being found insufficient, it was decided that an iridectomy must be tried; this was done on May 6, a large, free coloboma resulting. The eye did very well, healing quickly, the haze cleared and remained away almost entirely. Only once or twice, for short periods, had any dimness been manifested up to the time of leaving the "home," May 24, 1909. Soon after, the dimness began to recur as badly as ever, and the vision was rapidly declining, so subconjunctival injections of normal saline were tried with good temporary result, but in the course of a month or so this, too, failed to produce any benefit and it was stopped. By the end of 1909, vision Rt. only fingers at 10 feet and aided  $4/20$ . Tension + 1. Cornea clear; media clear; nerve deeply and widely cupped.

As, however, there were times when vision was clear and bright, it was decided, at the beginning of 1910, to try trephining the sclera, in accordance with the directions of Major Elliot. This was done on April 1, 1910, and the eye cleared up at once. The vision remained clear and good for nine days, when, the eye being quiet, the patient was allowed to be up. Next day the vision was a little cloudy, but soon became clear. A week later, the vision again became very dim and both surgeon and patient were much discouraged. The tension of the eye was slightly increased, even though the pad of œdema of the conjunctiva was present as markedly as at first. On April 30 the eye was becoming dim again and it occurred to me that gentle digital pressure on the globe at intervals might prevent the healing of the wound. This was tried with very satisfactory results. At the end of May, 1910, the eye continued quiet and fairly clear. Aided vision was now only  $\frac{4}{60}$ , but there was practically no dimness at any time. The pad of œdema continues as noticeable as ever. When last heard of, in April, 1912, the patient still maintained his satisfactory condition.

#### BUPHTHALMUS, OR INFANTILE GLAUCOMA: TREPHINING

The last case of glaucoma which I wish to bring forward is one of that class which is well known to resist treatment of all kinds ordinarily tried, namely, buphtalmus, or infantile glaucoma.

CASE IV.—The subject of this note, H. G. H., æt. 2 years, was brought to me in January, 1910. The history was that at the age of 6 weeks he had very severe whooping-cough and, after this, the parents noticed that the eyes were becoming larger and assuming a staring appearance, the right eye being the worse, but the left not very far behind it. The child was taken to an oculist at the age of 6 months, who diagnosed the disease as conjunctivitis and keratitis of strumous type, and atropine was ordered. The disease became worse and the child complained more, as if in pain. The enlargement of the eyes continued and the treatment was stopped by the parents. At the age of one year or so nystagmus affecting both eyes developed. Nothing was done for some time, the parents fearing that nothing could be done. When I saw the child, the eyes were very large, the right cornea measuring 15 mm. transversely and having a considerable central opacity. The eye was congested externally, the anterior chamber very deep, and the tension + 2. The left cornea measured 14 mm. and was slightly turbid and the anterior chamber was very deep. Tension of left + 1. The right eye retained P. L. only. The left eye could distinguish hand movements distinctly, but the patient was very shy and it was difficult to form a clear opinion regarding the vision. The child could not see to walk about in a strange place, but could at home. If any sight was to be preserved, something must be done at once and, as iridectomy held out little hope, it was decided to try Elliot's operation by trephining the sclero-corneal margin.

After a preliminary paracentesis of the cornea of the left eye, in order to ascertain the condition of the internal structures, the fundus was dimly seen, before the clouding of the cornea returned, and the nerve was found to be deeply cupped. Elliot's operation was successfully performed, as regards perforation of the globe, but I was not able to enter the anterior chamber, but got into

the vitreous at once, although the aperture was as far forward as possible. The disc excised, was examined histologically, and found to be "cornea with iris adherent to its posterior surface." At the end of a month the eye was quiet and the oedematous conjunctival pad manifest. The tension fell at once and the corneal turbidity disappeared. The eye has now, for two years, been at the worst stationary, so far as appearances are concerned. The tension seems to vary but little, usually being somewhat subnormal, and the oedematous pad of conjunctiva is still distinctly seen.

The parents considered the vision greatly improved, as the child could distinguish a dog on the road "a long way off" and recognized his father "coming up the garden," which he could not previously do. My own testing showed that the vision had improved, but it was difficult to say how much. The child was less shy than formerly and had developed in intelligence quite satisfactorily. When last seen the parents informed me that even such objects as telephone wires could be seen and I was able to prove, by throwing a thin wire (style wire) on the floor, that such objects could be seen but centrally only or nearly so. There is now no turbidity of the cornea, and, though the diameter is not less, it is not greater. The tension remains n. to — and the child can steer his way about quite easily and smartly, dodging chairs put in his way, etc. It has been noticeable for months that colors are correctly and easily seen, even shades of colors of flowers in the garden.

The right eye was operated on a year ago to see whether any benefit would result, as pain still continued and the cornea was more opaque than formerly. Although every effort was made to get the trephine opening far forward, hemorrhage into the eye followed, and the eye has shrivelled to a considerable extent.

It is probable that no more satisfactory result could be obtained in a case of this nature, especially when so far advanced, and one can only hope that the next case will do as well. It is certain that in such instances something must be done or total blindness will result and it is worth trying even so serious an intervention early rather than late.

#### THREE CASES OF EYE DISEASE TREATED BY TUBERCLE VACCINE

As vaccination by emulsions of organisms suitable for individual diseases promises to be of great service as a remedial agent, in some instances, at least, it is of interest to record the cases in which its use has proved successful, if not also those in which it has more or less distinctly failed.

As I have had two cases in which marked benefit followed the use of injections of emulsions of tubercle and one in which the benefit was too late to be of service in preserving sight, I think it well to put them on record now, after an interval of a year in the most recent case, and two and two and a half years in the others.

The possibilities in the matter of variety of vaccines is perhaps a little embarrassing to the surgeons. When the subject first came up, I studied the literature as well as possible and decided that of bacil-

lary emulsion was the most promising of the tubercle vaccines. I concluded to try the English rather than the German dosage. In all three cases English dosage was used and in the first two bacillary emulsion and in the third case T. R. new was the medium employed.

CASE V.—G. S., æt. 14, came to the eye infirmary in 1906, complaining of dimness of vision and pain in the right eye. The condition was found to be kerato-iritis and as the lad was badly developed and had numerous enlarged glands in various positions, it was considered probable that the cause was tubercle rather than syphilis. Various signs such as teeth, hair, nails, etc., pointed in the same direction.

The usual line of treatment, atropine ointment with syrup of the iodide of iron, was prescribed. The condition hung fire for a long time, without improvement, the pupil refusing to dilate fully and the cornea remaining cloudy. After about 6 months the lad was sent to the country, but did not do well there, an area of dense infiltration of the right cornea developing and the left cornea becoming hazy. He was again admitted to the infirmary and kept there for two months with distinct benefit; the right cornea cleared a little and the left eye became quiet. About six months later he returned to the out-door department in a worse condition than ever, both eyes being now involved to nearly equal extent; he was again admitted to the infirmary and once more improved. The visual power was greatly reduced at this time and he was sent to the country again for a period. After a lapse of some eight or nine months he returned to the infirmary, in his former condition, with both eyes involved. The pupils did not yield to atropine profusely used, the corneæ were very cloudy, and the pain was intense. It was then decided to try the effect of injections of B. E.,  $\frac{1}{10,000}$  mg. dose. This was given and no reaction followed; another dose was given ten days later with the same result, eight days later  $\frac{2}{10,000}$  mg. was given, a slight elevation of temperature resulting. The eyes began to clear up at this point; the photophobia and pain disappeared like magic, lachrymation ceased, and the conjunctival congestion diminished. The pupils, however, still refused to yield to any mydriatic. Ten days later another injection,  $\frac{2}{10,000}$  mg., was given, again without reaction, and after a week  $\frac{5}{10,000}$  mg. was given with only a slight reaction. Improvement in the local condition now became striking. The lad could see much better and went about the ward freely. Another injection of the same dose as the last was then given, no reaction following. The lad was sent home, to attend for treatment;  $\frac{8}{10,000}$  mg. was given twice at intervals of a week, and then  $\frac{1}{1,000}$  was given twice and, as the improvement was very striking, no further injection has been given.

The condition of matters was briefly this: The corneæ both cleared up wonderfully. The irides yielded a little at first and then more and more until at the present they are dilated to half maximum; the pupils are fairly clear, and the vision has improved slowly but steadily. The lad went to work on a farm, and he has never lost a day for the last three years on account of his health, which has continued to improve. Perhaps even more striking than the improvement in the eye condition was the improvement in the general development. The boy began to grow big and stout, and is immensely strong for a lad of his years. He is still working as a farm laborer, rises at 3 A.M. to milk the cows, etc., and seems to flourish on it.



His vision has gone on improving and when last seen (January, 1912), he could see  $\frac{6}{14}$  with the right and  $\frac{6}{10}$  with the left eye. All the injections, ten in number, were given in the arms, and each was followed a month or six weeks later by a "cold abscess," which healed quietly and left little or no mark. The glandular enlargements have very greatly diminished in size, only tiny nodules being palpable when last seen. It is now two and a half years since the last injection was given and no relapse has taken place since.

CASE VI.—The next case is that of R. M., æt. 30, a well-developed man whom I first saw early in December, 1910, on account of swelling of, and discharge from, the conjunctiva at the nasal side of the right eye. The history was that he never had trouble until eight months before, while in America, the right eye became sore and he consulted a local practitioner, who did everything he could for him. Glasses were ordered and lotion and ointment and other local applications used, but, in spite of everything, the eye grew worse; the swelling increased and "came to a head" and burst, discharging "clear watery looking matter." After subsiding a little, the swelling soon began again and he decided to come home. The voyage did him good, but he was not in this country long before the eye was as bad as ever. The condition was scleritis at the nasal and superior part of the right eye. Abscess formation evidently had taken place and a little discharge was still coming from an orifice in the thickening.

In the absence of history of rheumatism, syphilis or tubercle, I inclined to view the condition as tubercular, as absence of pain was striking and the discharge was of a watery albuminous appearance such as I had seen before in a somewhat similar condition. Careful urinalysis discovered the presence of oxalates in abundance; the ratio between uric acid and urea was good, indicating freedom from gouty disease. As the patient strongly maintained that there was no evidence of tuberculosis in his family or himself, I consented to try local applications for a month or two, but, while this and general tonics seemed of benefit at first, it soon became manifest that this was only an interval in the disease, which returned as bad as ever. I then insisted on using a vaccine, and administered  $\frac{1}{10,000}$  mg. dose of T. R. *new* in the lumbar region; no reaction followed, and  $\frac{2^{16}}{10,000}$  was given a week later. The eye cleared up in a very striking manner during the week after this second injection and the abscess dried up and never broke out again. In order to be sure, the patient was kept for a week, and an injection of  $\frac{3}{10,000}$  mg. was given. No reaction followed this injection, and the eye being quite white and clear, he was sent to the country for a month. He stayed nearly two months and returned perfectly well, without any signs of the former condition. The conjunctiva was clear of injection and no scar was visible. He remained in town another month and then went back to America, having given me a promise that, if anything went wrong, I would hear from him. I have had no word during nearly a year and conclude that he has remained well.

No cold abscesses formed in this case and not the slightest inconvenience was experienced as a result of the injections.

The next case is one in which, greatly to my vexation and disappointment, the use of vaccine treatment failed to bring about cessation of the disease, and is one from which I have learned much. As the treatment extends over six years it cannot be compressed into small



compass, and I feel that to record it very briefly would be to lose much of its value.

CASE VII.—A. M., æt. 20. When I first saw the patient, in March, 1905, she was a strong, healthy-looking girl, and was employed in a dairy at book-keeping and counter work. Both her parents are known to me as healthy, active people, the father spare and vigorous for his years, the mother stout and bright. They are a family of five, all well in 1905, except A. M. herself.

The history was that in 1903 her eyes became sore for a time, but recovered perfectly. In 1904 some inflammatory troubles of the eyes again occurred and a "mist" came over the right eye. She was under treatment this time and recovered fairly well, the eye being nearly as "good as ever." No trace could be had of the name given to the disease at this time. This state was maintained until the end of January, 1905, when a "mist" came over both eyes without any attendant redness or pain. She was again under treatment and this time it was said that "keratitis was the disease." When I first saw her in March, 1905, she had been under treatment for six weeks, but was getting worse. V. A. R.  $\frac{16}{200}$ , L.  $\frac{16}{40}$ . The right cornea was steamy and when examined by strong light and a magnifier, revealed large numbers of small rusty-colored dots on its posterior surface, mostly at the lower part; the same state, though much less marked, was seen in the left cornea. The fundus of each eye could be seen only very dimly, but the retinal vessels, especially the veins, were found to be distended and tortuous. The tension of the right eye was — and of the left eye +. The anterior chamber was of normal depth, the iris being dull and discolored but dilating fairly well to atropine, which had been used freely. The general state was apparently excellent. There were no enlarged glands anywhere; the patient was, and is still, cheerful and bright.

The diagnosis was "serous cyclitis" and appropriate remedies were prescribed. No benefit resulted, the sight became worse till on March 25, 1905, the right eye could only distinguish large moving objects and the left  $\frac{16}{200}$ . This diminution of vision was principally due to steamy haze of the cornea which I concluded was due to œdema. Tension as before, right —, left +. On April 1, 1905, the condition suddenly improved. Right, V. A.  $\frac{16}{70}$ , left,  $\frac{16}{30}$ ; corneæ brighter. On April 8, left eye was worse. Cornea steamy. V. A. R.  $\frac{16}{40}$ , L.  $\frac{16}{200}$  dimly. Both eyes got worse for ten days till the patient could no longer see to go about. On May 10, the eyes had improved greatly, the corneæ being now clear, and vision was much better. V. A. R.  $\frac{16}{36}$ , L.  $\frac{16}{200}$ .

By May 24 both eyes were improved, the cloudy appearance of the cornea being absent; V. A. R.  $\frac{16}{30}$ , L.  $\frac{16}{25}$ . Spots of deposit on Descemet's membrane were visible in both eyes. Early in June, 1905, the right eye relapsed, but by the 16th had cleared up and no further relapse took place till December, 1905, when the right eye was again bad for a fortnight. On December 15, 1905, the corneæ were clear and vision right =  $\frac{16}{20}$  left =  $\frac{16}{25}$ . A few spots were seen with the magnifier on the posterior surface of each cornea. The eyes remained well till December, 1906, and the woman returned to work for nearly a year. Early in January, 1907, she came to me again, the right eye having been again bad and for nearly six weeks had not cleared up at all. The left eye became dim, but had cleared up and relapsed twice during this time. There was acute pain now. T. R. and L. + 1. Corneal very steamy; pupils medium in size. V. A. R. and L.,

large objects only, and the patient admitted to the infirmary. It was felt that paracentesis should be tried, and this was done and repeated with the result that the corneæ cleared up for a time but relapsed. Then an iridectomy was made in each eye; healing was slow but quite satisfactory. The pain disappeared a few days after operation and the corneæ cleared up. V. A. R.  $\frac{3}{200}$ ; V. A. L.  $\frac{15}{20}$ , with corrections for astigmatism a month after the operation.

A month later, after a good interval, both eyes became very bad again and subconjunctival injections of normal saline solution were tried with excellent results, temporarily; the vision cleared up to  $\frac{15}{80}$  right,  $\frac{15}{45}$  left. The eyes remained quiet for a few months and then the right one again relapsed for a time and on June 1, 1907, V. A. R.  $\frac{15}{200}$ , L.  $\frac{15}{20}$ , fairly clearly. The right eye did not clear up again from this point. Hypopyon developed and the iris became thickened in appearance, while nodules developed in the cornea which, I felt sure, were tubercular in nature. The condition remained much the same for some weeks, the left eye remaining clear and the right irritable, with small hypopyon and nodules in the cornea.

By September, 1907, the vision of the right had improved to fingers at 7 feet, the left remaining very good,  $\frac{15}{17}$ . There was no pus in Rt. A. C. now but a small hyphæma. Dust-like opacities were now, for the first time, detected in the vitreous of the left eye. For another month the eyes remained quiet and corrected vision of left was  $\frac{15}{11}$  on October 7. It was decided to try Calmette's serum diagnosis and this gave a distinctly positive reaction. The right eye remained in a dull, quiet state, tension  $\pm$  or  $\pm 1$ ; cornea cloudy in places; scleral vessels injected; no pain, V.  $\frac{3}{200}$  only. The left eye retained its vision  $\frac{15}{12}$  till December, 1907, when an attack of dimness with pain occurred. The right eye remained quiet, the left cleared up well and remained quiet for nearly nine months. Vision, August 8, 1908, right  $\frac{9}{200}$ , left  $\frac{20}{20}$ , aided. At this time it was noted that a gland in the left side of the neck had become enlarged and many other enlarged glands were felt: the teeth also were found to be somewhat defective. A brother had taken ill about a month previous with pleuro-pneumonia, and it may be said here that, after a long illness, he died of phthisis pulmonalis. This illness had affected the patient, A. M., very much, and the left eye again became bad in September, 1908. After treatment in hospital it cleared up so that by December 21, 1908, the vision was  $\frac{15}{25}$  aided. Still cloudy dust-like opacities in the vitreous of left; T  $\pm$ ? or n. at times. This state was maintained until May, 1909, when a slight attack of dimness began in the left eye, but cleared up well and remained so till August, 1909, when it returned. It was then decided to give vaccine treatment a trial, and six injections of B. E.  $\frac{1}{10,000}$ , gradually increasing strength, were used without reaction or visible result, so far as the eyes were concerned. The vision of the left eye remained very dull,  $\frac{15}{100}$  to  $\frac{15}{200}$ , till the beginning of November, 1909, when a slight improvement set in, but was not maintained for very long. The eye remained quiet; no pain, no redness, but the vision gradually diminished and cataract developed in both eyes, so that P. L. was all that remained.

This patient has been seen at intervals and no further acute attacks have occurred. When last seen, February, 1912, V. = P. L. and fair projection in left P. L. only in right. Tension right and left  $\pm$ . Irides discolored and cicatricial; marking nebulous cornea especially marginal, both eyes. Slight tendency to bulging at the upper part of each, right worse. There is opacity of the lenses

of an unhealthy looking type, the anterior part of the lens looks fairly clear, but there is a patchy opacity in the central and posterior parts. The general health and spirits are in every way excellent. The patient is learning to read and write Braille and is able to do housework. There has been no pain nor redness of the eyes now for a year, and the enlarged glands have almost disappeared. The question of removal of the cataract must remain *sub judice* for a year or two, as to touch such eyes would be to court disaster.

One wonders whether vaccine treatment was used too late, or whether it was not suited to this particular case, or whether bacillary emulsion would not have been the proper thing. All kinds of treatment, medicinal and others, were tried, but neither iron, iodide, mercury, salines, nor opium gave permanent results. I fancy that, had I the same sort of case to treat again, I would try iridectomy at an earlier stage; but the wonderful way in which the eyes cleared up after an attack always gave hope of a cure, and it is difficult to say how to regard the matter. Certainly, I would try vaccine treatment, possibly not bacillary emulsion, but T. R. new, and in small doses, as in the present case. The largest dose given was  $\frac{5}{10,000}$  mg., but twice that amount might have been used if the least benefit had appeared to result.

It is possible that had vaccine treatment not been used the result might have been even worse, and if the disease had been tubercular in nature it would most likely have progressed. This is certainly the only case of serous cyclitis of this type which I have seen, but the case of A. T. L., described above under glaucoma, resembles it somewhat.

# Obstetrics and Gynæcology

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## X-RAY IN OBSTETRICS: RADIOGRAPHIC DIAGNOSIS OF PREGNANCIES

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CHICAGO

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X-RAY skiagraphs will now demonstrate the fœtus *in utero* as early as the fourth month, and the stereoscopic radiographs will show the shape and size of the pelvis, thereby helping very considerably in diagnosing the possibility of mechanical difficulties in labor, gross deformities, distortions, and narrowing of the pelvic outlet, also the larger portions of the lumen of the pelvis, and decide the question as to which side of the pelvis the large occiput of the head should be brought down for delivery.

I will discuss briefly the question of the radiographic diagnosis of pregnancies, dividing the subject into two parts:

First: The fœtus *in utero*.

Second: Abnormal conditions in the mother.

### THE FŒTUS IN UTERO

The fœtus *in utero* may be detected from the fourth month. I should hesitate to say that any abnormality of the fœtus could be determined at that stage, but the position of the fœtus and any abnormalities of the uterus can be plainly seen. Twins could be determined, and in the question of illegitimacy, when an abdominal examination could not be suggested or permitted, the X-ray would discover the fœtus and make diagnosis positive.

I had a case of a young girl, aged 18 years, suffering from dementia præcox, brought by her mother, aged about 40, also with mental symptoms, both accusing the father, a rather elderly man, of rape, and on their testimony he was confined in the county jail for about four months, both mother and daughter insisting upon pregnancy. This was indorsed after a superficial examination by a physician. I made

a radiograph of the girl—at that time she would presumably be about five months pregnant—but found the uterus normal. Showing the mother the pictures taken of her daughter, also some radiographs with the fœtus *in utero* of about the same length of time, she confessed before two medical men, who were visiting in my office, that it was a put-up business to get the father out of the way, to secure during his detention in jail his insurance policies and other small assets. In this case abdominal examination was refused, and the X-ray consented to because advice had already been obtained from another physician that the fœtus could not be shown by the X-ray. The man was liberated on the evidence of the radiograph.

After the sixth month, gross fetal deformities, such as hydrocephalus and anencephalus, double monsters, etc., would be readily discernible, and up to the last week of term a skiagraph would, in most cases, be of great value.

The X-ray would be valuable in early infancy. By its means hyperossification, due to prolonged pregnancy, possibly could be determined. Injuries, such as fracture of the skull, fracture of the extremities, etc., and one might find a hemorrhage in the brain by its means.

#### ABNORMAL CONDITIONS IN THE MOTHER

An extra-uterine or tubular pregnancy could not be diagnosed as such. The diagnosis between a normal pregnancy and hydatid mole, a lithopedium, or calcified fibroid would show well, and before every cæsarian section it would be of infinite value both for determining the pelvic shape and condition of the mother and the position of the fœtus, and any abnormalities, if present. After a rupture of the pubic joint a series of skiagraphs taken during the healing processes would be of great value to the obstetrician.

The differential diagnosis between rupture of the pubis and softening and relaxation of the pelvic joints could be made by this means, and the radiograph would determine between polyneuritis, paraplegia, and pelvic bone disease.

#### TECHNIQUE

In the hands of an expert radiologist, and since modern technique requires only a slight fraction of a second exposure to both mother



FIG. 1.



Patient lying in position as described on p. 269. The anode of the tube should be 40 inches from the sensitive plate. A small metal button should be placed on the umbilicus.

FIG. 2



Patient lying laterally, with spine of fetus in utero nearest to sensitive plate.

and child, it is rendered safe and free from injurious effects. The technique of making the radiograph varies with the length of term, weight, etc., but approximately the following *modus operandi* should be followed:

An interrupterless transformer or a standard coil of any good make works equally well. I am using a 4 KW. Scheidel Western interruptless transformer. The tube should be very soft and should start on about 3 milliamperes of current.

For about a four-month pregnancy, 25 milliamperes of current should be flashed for about one-fourth of a second; that is, for a subject of average weight, 135 pounds, and about 4 to 5 months' duration. For each succeeding month and 10 pounds more weight, one-sixth of a second more exposure should be given.

The patient is prepared by first having the bowels thoroughly cleaned out with suitable cathartic and enemas, the back and whole abdomen rubbed lightly over with liquid petrolatum. This is not necessary, but I have found in some instances that even in these quick exposures the patient has complained of a slight irritation after exposure—I think imaginative in most cases—possibly due to the distended abdomen being more sensitive, but have had no complaint when the petrolatum had been used.

It is best to make four pictures of each subject, care being taken to protect her from all exposure to draughts, chills, etc. The first two should be taken with the patient lying on the abdomen with body curved, as in Fig. 1. The knees and chest should be propped up with suitable cushions or pads, so that there is no pressure, and the belly of the subject just touching the plate. The umbilicus should be in the centre of a 14 x 17 plate.

The other two plates, taken laterally as in Fig. 2, should be exposed after the first one has been developed, so that some idea of the position of the fœtus may be obtained. The spine of the fœtus *in utero* should rest nearest to the sensitive plate.

As an example of this kind of work the reader is referred to Figs. 3, 4, and 5.

The development of the plates should be very slow, depending upon the make. The ones I find most suitable and slow are those manufactured by the Cramer Dry Plate Company. Very rapid plates should not be used, as they lack detail in the bony structure of the fœtus. I find the best method of development is in three solutions, the formulæ of which are:

*Solution A*

Hydrochinon .....	1 drachm
Sodium sulphite .....	1¼ ounces
Sodium carbonate .....	1 ounce
Potassium bromide .....	2 drachms
Water .....	84 ounces

*Solution B*

Metol .....	60 grains
Hydrochinon .....	3½ drachms
Sodium sulphite .....	3 ounces
Sodium carbonate .....	3 ounces
Potassium bromide .....	3 drachms
Water .....	64 ounces

Three trays, perfectly clean, should be used, containing:

Tray No. 1—Solution A.

Tray No. 2—Solution B, half strength (developer 1 part, water 1 part).

Tray No. 3—Solution B, full strength.

The plate should be taken out of the envelope and dusted with a large camel's-hair brush, and immersed in Solution A for about 4 or 5 minutes with continuous rocking, then taken out and immersed in Solution B for at least 10 minutes or longer, or until the vertebra and pelvis of the mother may be faintly seen, and then immersed in the full-strength solution until all detail is brought out. Failure to obtain good negatives is usually due to overexposure or hasty development. If developed slowly and in 3-strength solutions, as advised above, the hard parts are retarded, giving more equal density to the negatives.

As the development is somewhat long, the first part in Solution A and the second part in Solution B should take place in absolute darkness, and only the ruby light turned on during the third development. Weak plates, due to underexposure, can be intensified, but generally lack so much detail that they are valueless.

FIG. 2.



Intestinal outlines, showing appendix.



FIG. 4.



Ossified foetus, possibly of four years' duration.

FIG. 5.



*Normal fœtus in uterus at six and one-half months.*



## THE RECOGNITION AND TREATMENT OF THE COMPLICATIONS OF GONORRHŒA IN WOMEN\*

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IN two preceding articles<sup>1</sup> the manifestations of gonococcal invasion of the lower female urogenital tract have been considered. While involvement of a region other than the urethra, bladder, cervix, and uterine cavity is usually caused by extension of the primary infection from one of these localities, and is not a true "complication," such conditions are ordinarily viewed as complications because they are due to either virulent infections, dyscrasia, neglect, or carelessness. Prompt recognition and appropriate treatment of the primary genital infection are of considerable importance, since the longer a gonorrhœa exists the more chronic it becomes, and a woman so afflicted is a constant menace to herself, family, and friends. However, while the control of early gonorrhœa in women is essential for hygienic, social, and economic reasons, the recognition and rational treatment of the so-called complications are not only of the utmost consequence, but absolutely essential, since these conditions jeopardize life.

GONORRHŒAL METRITIS, OR MYOMETRITIS.—This is a gonococcal inflammation of all the cervical and corporeal structures of the uterus, and is invariably acute. The infection extends by continuity of surface, rather than by the lymphatics and veins, as in metritis due to etiological factors other than the gonococcus. It is characterized by initial chill, suprapubic pain, manifestations of bladder irritation, elevation of pulse-rate, pyrexia, and nausea. There are swelling, tenderness, and œdematous softening of the uterus, physical signs easily detected by bimanual examination. The inflammation and its products usually extend to the adnexal structures, particularly the broad ligaments (parametritis), and there may be sufficient exudate to produce an indefinite mass of board-like consistency on each

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side of the uterus. A pyosalpinx or pelvic abscess, however, would obscure the latter evidence.

*Treatment.*—Prophylaxis, such as clean technic in pelvic examinations and manipulations, and proper treatment of the primary foci, as a preventive against the development of all complications, is assumed, provided the physician has been afforded the opportunity to control the case from its inception. As a matter of fact, the patient frequently attempts to care for herself and neglects to consult her physician until the infection has progressed extensively, so that complications are often present when she is first examined.

The immediate treatment of gonorrhœal metritis and parametritis may be summed up in two words: (1) rest and (2) depletion, of both the uterus and general economy. All intra-uterine intervention, especially routine douching and curetting, is absolutely contra-indicated. The patient should be kept in bed and the diet limited to liquids. This applies to all cases of acute pelvic inflammation due to gonococci, irrespective of the anatomical location. The application of an ice-bag over the hypogastrium serves to allay inflammation and pain and limit the exudate, as well as to prevent uterine tenesmus. If the pain is violent, rectal suppositories containing one grain of opium are permissible.

Hot fomentations on the abdomen are not employed, as they tend to promote purulent resolution of the exudate. Hot vaginal douches, on the contrary, are useful, since they increase the pelvic leucocytosis. They should be administered in large quantities at frequent intervals. Saline solution, a saturated solution of boric acid, or one drachm of the tincture of iodine to two quarts of water are satisfactory agents for this purpose.

Simple saline cathartics stimulate the activity of the emunctories and thus facilitate general depletion. One drachm of sodium phosphate may be given in vichy each morning. In severe cases accompanied by hyperpyrexia an enema of saline solution containing four ounces of magnesium sulphate and given at 70° F., repeated every four hours if necessary, will prove efficient. Such a colonic irrigation will aid in the elimination of toxins and reduce the fever.

As the acute inflammation subsides, vaginal tampons of iodine-glycerine 10 per cent. or boroglyceride may be inserted on alternate days, to promote uterine depletion. When the myometritis has



entirely abated, the muscle may be disregarded and the case treated as one of endometritis. Curettage and appropriate intra-uterine after-treatment are then indicated.

GONORRHOEAL SALPINGITIS.—This affection nearly always exists as a suppurative inflammation, and may be acute or chronic. The chronic type is probably more often encountered than the acute, and since the latter usually merges into a pyosalpinx, because the sudden inflammatory swelling obstructs drainage, it will be disregarded here and the chronic form only considered.

A correct conception of the pathological condition in each case is of the utmost importance, as the method of treatment selected should depend upon a proper interpretation of the local findings. All the coats of the tubes are involved and the ciliated epithelium is invariably destroyed. The outer ends of the tubes are usually sealed, but sometimes remain patent. Exacerbations of local peritonitis may occur from leakage, hence the importance of the patients avoiding undue exertion and traumatism. The pelvic organs are often agglutinated by adhesions, the vermiform appendix and ovaries being frequently involved. The termination may be either resolution or a seropyosalpinx. Many cases are entirely resistant to leucocytosis and all palliative measures, and extirpation is the only alternative. Needless to say, a tube constantly discharging gonorrhœal material is an absolute barrier to the cure of a coexisting gonorrhœal endometritis and a relative impediment to pregnancy. Should conception occur thereafter, extra-uterine pregnancy is likely. However, sterility is the rule.

The manifestations of a gonorrhœal salpingitis are: antecedent history or presence of primary infection, leucorrhœa, local pain and tenderness, dysmenorrhœa, dyspareunia, recurring attacks of localized peritonitis, and some systemic disturbance accompanied by elevation of pulse-rate and temperature (varying in direct proportion to the virulence). As the condition becomes more chronic the symptoms gradually abate. Complete spontaneous cure is uncommon. Occasionally the fimbriated extremity, previously open, becomes sealed, due to excess of exudate at that point, with blocking of the uterine end by swelling, so that a pyosalpinx ensues.

On examination, the tube is found thickened and sensitive and surrounded by inflammatory products and adhesions. The mobility

of the uterus is generally limited, this organ being agglutinated with the tube and corresponding ovary. Sometimes the parametritis and local peritonitis may be sufficiently severe to undergo suppurative changes, and pelvic abscess results. A gonorrhœal endometritis that persistently resists all proper remedial measures, particularly if accompanied by a mucopurulent nontenacious leucorrhœa, should arouse suspicion of an associated salpingitis.

*Treatment.*—The rational treatment must be based upon the chronicity, extent of involvement, virulence, and individual factors. In the more recent cases vigorous treatment is contra-indicated. Hot vaginal douches of iodine solution, two or three gallons twice daily, suitable tampons, and meeting symptomatic requirements are more logical. Later, cautious pelvic massage, kneading the tube from its fimbria toward the uterus, repair of existing cervical lacerations, and curettage of the uterus often cure. When curetting, the operator must be careful not to drag down an adherent uterus forcibly, owing to the likelihood of exciting an inflammatory exacerbation in the adnexa. Although it has been claimed that the fallopian tubes may be catheterized through the uterus and drainage maintained thereby, the writer has demonstrated, as have many others, that such manipulations are quite impossible, even in extirpated organs. When the uterine extremity is not too greatly constricted, however, prolonged uterine drainage will promote drainage of the tube. In those cases becoming progressively worse, or those in which all palliative measures fail, salpingectomy should be performed. This need not necessarily be complete in every instance, as the diseased portion of the tube may be excised and the corresponding tunics of the stump approximated with absorbable sutures. Without being radically conservative, all tissue not hopelessly diseased should be preserved.

**GONORRHOËAL PYOSALPINX.**—Gonorrhœal pyosalpinx may begin as such almost simultaneously with the uterine infection, if the latter is particularly virulent, or may occur as the termination of a chronic gonorrhœal salpingitis. The former would probably occur as an acute pyosalpinx, the latter chronic. In all cases of pyosalpinx both the uterine and abdominal ends of the tube are closed and the tube is distended by purulent accumulation. In acute cases the bacteria are active and the infection is virulent. On the contrary, in chronic cases the microorganisms are usually dead and the pus sterile.

The tube is often adherent to the peritoneal cul-de-sac, uterus, ovary, omentum, intestine, or appendix. The pus-cavity may rupture and a local abscess or general peritonitis result.

The symptoms in acute cases are usually pronounced, being all those of a highly active inflammation, and are aggravated on standing, walking, or other exertion. Elevation of the pulse and temperature varies in direct proportion to the activity of the infection, and is a reliable guide thereto. Systemic disturbances are marked. In chronic cases the symptoms are not necessarily prominent, although recurrent attacks of pelvic peritonitis frequently occur at the menstrual periods. Leucorrhœa is relatively constant. The tube is thickened and tender, and located at the side of or behind the uterus. The size of the tumor may vary from that of a finger to that of the fist. Uterine mobility is limited or lost and attempts to move the uterus cause pain. Fluctuation is often difficult to detect on examination. In the presence of many adhesions, palpation of the pelvic structure is impossible, and the tube may be indistinguishable from the general mass.

*Treatment.*—The treatment will depend upon the activity of the germs and the extent of suppuration. In general, it may be said that an acute pyosalpinx should never be operated upon immediately, whereas in all chronic cases in which the tube is destroyed the latter should be removed. Efforts to cure an acute pyosalpinx entirely are usually futile, and palliative measures should therefore be directed toward lessening its activity and virulence, converting the acute process into one of the chronic type. The rational treatment of cases characterized by the manifestations of acute inflammation, particularly by marked rise in pulse-rate, temperature, and leucocytosis, is essentially that of acute salpingitis. Operative measures other than vaginal evacuation in event of abscess formation, especially intra-uterine interference, are extremely dangerous, because of the likelihood of infecting the peritoneum with the intensely virulent pus. Should this occur, general gonorrhœal peritonitis rapidly ensues, the mortality of which is high. It is wise, therefore, to confine the initial treatment to such measures as will alleviate the acute inflammation and symptoms.

Complete rest in bed, saline catharsis, cold applications on the abdomen, frequent hot douches, and a light diet should be continued

until the temperature and pulse-rate both fall below 100 and remain there for twenty-four hours, when operation is reasonably safe. But should this not occur after the lapse of one week, and the evening temperature approximates 102° F. or under on two or three successive days, operation is advisable. Also, the hyperacute cases that become progressively worse, in spite of all efforts to control them, require immediate operation.

Except in those acute cases in which the pus-tube lies in the cul-de-sac and bulges into the posterior vaginal fornix, laparotomy is the preferable method of operating. In the former instance, however, free vaginal incision, evacuation, and drainage, as for pelvic abscess, are not only judicious but will result in cure without additional measures in 50 per cent. of such cases. This practically eliminates all possibility of contaminating the peritoneum with the infectious pus and maintains satisfactory drainage, which is practically extraperitoneal. On the contrary, in all other recent infections (interval operations) and all cases of long standing, anterior or posterior vaginal section will only hamper the operator, without offering any material advantages. The field for manipulations is limited, thorough exploration of the lower abdomen impossible, and technic necessarily uncertain. So, to permit free inspection of the appendix, lower bowel, and adnexa, separation of adhesions, extirpation of all diseased tissue, and proper treatment of associated pathological conditions, abdominal section is the method of choice. The tube itself may be incised and drained, probed, resected or removed, according to the individual case and the surgeon's judgment. Few cases will require subsequent peritoneal drainage, even though the tube is ruptured during operation, except when there has been an acute exacerbation in a chronic case or in presence of a concomitant pelvic abscess. When drainage is necessary, that through a stretched stab-wound in the cul-de-sac is preferable, because of its being the lowest level of the abdominal cavity.

Gauze or rubber tubing may be utilized for drainage, although the writer never employs the former. But when gauze is selected, it should be left in place for two or three days and then all removed at once. Nitrous oxide gas may be administered, if necessary, to facilitate removal. When rubber tubing is inserted, it is more satisfactory to use two tubes stitched together than one alone. Irrigation



is then easy, the fluid entering through one tube and returning through the other. Obviously, the Fowler position should be maintained in these cases, so that gravity will aid drainage to and through the pelvis. The writer relies entirely upon iodine for hand sterilization and employs iodine catgut exclusively as suture material in all pelvic and abdominal operations.<sup>2</sup>

**GONORRHOEAL PELVIC CELLULITIS AND ABSCESS.**—These conditions are invariably the result of extension from the pelvic viscera and peritoneum, and are never primary infections. Cellulitis and abscess are nearly always accompanied by some degree of peritonitis, and conversely. The cellular tissue at the base of the broad ligaments, where it is most abundant, is the most common location of the inflammation, and this is usually termed parametritis. Cellulitis caused by gonococci rapidly undergoes purulent resolution and abscess follows. This most often bulges into the posterior vaginal fornix, but may point elsewhere and rupture into the bladder, vagina, or rectum. Pyosalpinx, ovarian abscess, and pelvic neoplasms must be excluded. Local tenderness, tumefaction, bogginess, and uterine fixation are characteristic.

*Treatment.*—Palliative measures, such as applications of heat or cold, douching, tampons, etc., are usually of no avail in event of abscess formation. The cul-de-sac should be incised and drained promptly. Laparotomy is unnecessary and often dangerous. Should the pus rupture into the bladder or rectum, a vaginal counter-opening should be made in addition.

**GONORRHOEAL OVARIAN ABSCESS.**—This affection is due to partial destruction and suppuration of the ovary following acute oöphoritis and peri-oöphoritis. The concomitant adhesions limit the destruction of tissue somewhat and serve to protect the general peritoneum from invasion. This condition is easily recognized, as the ovarian pain and tenderness are intense, the ovary is markedly enlarged, and the abscess can nearly always be detected on bimanual examination.

The treatment is simple. The abdomen should be opened, the peritoneum and abdominal viscera protected by walling off with large gauze pads, the abscess ruptured, and the pus rapidly mopped out. Other structures diseased are resected or treated otherwise appropriately, and the peritoneal cavity drained through a stab-



wound into the posterior vaginal fornix. Attempting to remove the pus by intra-abdominal irrigation is a dangerous procedure, because in spite of all precautions the infection will almost invariably be disseminated in the peritoneal cavity. Dry operating (as far as possible) affords infinitely better results.

**GONORRHEAL PERITONITIS.**—In adults this disease is generally limited to the pelvis and most frequently proceeds from infected tubes. In the acute form the symptoms and physical signs do not differ from those of a localized peritonitis elsewhere. Chills, pyrexia, increasing rapidity of the pulse, pain, tenderness, muscular rigidity, tympanites, fixity of the pelvic organs, and gastro-intestinal disturbances are all characteristic. In chronic cases the manifestations are not so pronounced, except in event of acute exacerbations, which, as a matter of fact, are the rule. Here there is but little pain, fever, or tenderness. The pelvic viscera are immobile and agglutinated by adhesions. Encysted serous or purulent accumulations are not uncommon.

*Treatment.*—In acute septic cases the treatment should be palliative and more or less expectant, unless it is evident that the infection is steadily progressing. Complete rest in bed, ice-bags on the lower abdomen, stimulation, free catharsis, and diuresis are advisable until the acute process subsides or it becomes evident that the infection is hopelessly beyond control. When laparotomy becomes imperative, an individual case may require anything from simple pelvic drainage through the cul-de-sac to a panhysterectomy with removal of the tubes and ovaries. No fixed rule can be promulgated in advance for the operative treatment of all cases. The operator must exercise his judgment and discretion in dealing with conditions as he finds them.

**SYSTEMIC GONORRHEAL INFECTIONS** are far more common than recognized. Anorexia, gastro-intestinal disturbances, slight elevation of temperature, leucocytosis, and the other indefinite deviations from the normal observed in nearly all cases of fresh gonococcal infections, even when limited to the external genitals and endometrium, are manifestations of a certain amount of absorption of poisonous material. While usually not of sufficient intensity to demand serious consideration, yet cases are occasionally encountered showing profound intoxication, in which the elaboration and absorption of toxins is more rapid than their disposal. These have been classified

by Pearce<sup>3</sup> as: (1) gonococcal toxæmia, (2) gonococcal septicæmia, and (3) gonococcal pyæmia. Thus, the intoxication may be due to (1) the entrance of gonococci into the circulation, with or without the formation of metastatic abscesses, or (2) overwhelming the patient's resistance by absorption of the toxins elaborated by the gonococci, without the microorganisms passing into the bloodstream. De Christmas's<sup>4</sup> experiments on rabbits and guinea-pigs proved conclusively the presence of a virulent gonotoxin in every gonorrhœal infection. Severe gonorrhœal sepsis is more often observed in females than males, although not of great frequency in either sex. Townsend and Valentine<sup>5</sup> have recently published a brief consideration of the subject and cite an illustrative case, typical of gonococcal toxæmia.

*Treatment* consists of (1) removal of the source of absorption, and disinfection of what cannot be removed, (2) promotion of elimination of toxins, and (3) combating the constitutional symptoms by supporting the patient's vitality.

Appropriate local measures and disinfection of all the primary foci usually suffice to cause the disappearance of toxic symptoms, by arresting the propagation of gonococci. The excretory organs must be kept active to stimulate elimination. Physiologic saline solution, given in enemata or intravenously, dilutes the toxins and acts as a circulatory stimulant. Most satisfactory results will be derived from enemata, each consisting of four ounces of magnesium sulphate dissolved in a pint of cool water (70° F.), repeated every four hours until improvement is pronounced. This hypertonic solution reduces fever, causes copious watery stools, and promotes rapid disposal of toxins by osmosis through the intestinal mucous membrane. The use of autogenous vaccines or serums is not practicable in these cases, because the attack is usually acute and of short duration, allowing no time for their manufacture. The results obtained from the use of stock preparations have not been encouraging. In fact, vaccines have disappointed in all gonorrhœal cases except those of joint lesions.

**OTHER COMPLICATIONS.**—Lack of space precludes discussion of some of the other complications. Among these may be mentioned gonorrhœal infections of the kidney, arthritis, endocarditis, and ophthalmia. It seems wise to recall, however, that while the gono-

coccus may not be isolated in puerperal sepsis, the presence of gonococci during pregnancy certainly predisposes to streptococcic and mixed infections after labor.

THE COMPLEMENT-FIXATION TEST IN THE RECOGNITION OF GONOCOCCIC INFECTIONS.—Schwartz and McNeil<sup>6</sup> experimented with the complement-fixation test in 324 cases, using a multivalent antigen prepared from many strains of gonococci. They found that this antigen fixes complement whenever one of its component strains does so. While they admit that a negative result will be obtained in a positive case if the strain in that particular case differs markedly from any present in the polyvalent antigen, they assert that a positive reaction will be obtained in the majority of cases that harbor gonococci. In other words, these results seem to prove that a positive reaction is indicative of living gonococci somewhere in the body, and when detected in patients suspected of having had a gonorrhœal infection their capacity of infecting others should be assumed. Differential diagnosis and a diagnosis of chronic gonorrhœa are therefore reasonably sure.

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# Occupational Diseases

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## OCCUPATIONAL HYGIENE IN THE NAVY

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UNDER the great subject of hygiene, the division comprising the prevention of pathological states arising directly from conditions dependent upon certain occupations has become one of tremendous interest and importance the world over. This has been fully realized in the navy of the United States, and by careful and intelligent study of statistics and the able and constant observations of its medical officers it has been possible accurately to locate the points of attack, and to make provision for their betterment.

Occupational diseases in the navy may be divided into two great classes: (*a*) Diseases existing to a greater or less extent among civilians which become occupational by reason of conditions inseparable from life in a military establishment; and (*b*) those directly and solely resulting from the peculiar nature of the duties performed in the naval service.

Under class (*a*) may be grouped general infective, skin, and venereal and non-venereal diseases of the genito-urinary tract, lead poisoning, and flat-foot. Under class (*b*) occur eye-strain, effect of gunfire upon the ears, heat exhaustion and thermic myalgia, caisson disease, the effects of powder gases, and certain accidents, pre-eminent among which is drowning.

The general infective, skin, and venereal and non-venereal diseases become occupational in the navy largely through the close association of large bodies of men on board ship and in barracks, intensified, in the case of recruits, by youth and sudden change in environment. The influence of the latter element is graphically shown in a statistical table of diseases based upon occupational groups, which appeared in the Annual Report of the Surgeon-General for 1911. For example: Among midshipmen and apprentice seamen, the two

groups of the navy personnel which are distinctly composed of those new in the service, the admission rate per thousand for contagious diseases for the year ending June 30, 1910, was 417 and 598, respectively, while the average rate for all other groups was 84, and for the one next below midshipmen on the scale it was only 123 per thousand. To meet this condition detention barracks have been established at the large recruiting depots where the newcomers are isolated in small units for a period of twenty-one days; isolation rooms form a structural part of the new vessels; methods of disinfection, both steam and chemical, are available; ample facilities for bathing and scrubbing clothes are provided (shower baths have been substituted for buckets and tubs); the most modern toilet facilities have been installed; the sanitary bubbling-spring faucet has replaced the old, universal drinking cup; and extensive screenings have been installed to limit fly- and mosquito-borne diseases. In addition, the men are trained to care for their persons and effects under the new conditions; they receive instruction in the dangers of venereal disease and are provided with prophylaxis; they are constantly inspected, and the use of prophylactic inoculation for smallpox and typhoid fever is compulsory. This will serve to indicate something of what is being done to limit the damage caused by diseases of this class.

Tuberculosis in the navy, as in civil life, has received careful study. During the year ending June 30, 1910, this disease was the cause of 19 deaths and 11,913 sick days, excluding all cases under treatment at the Naval Sanitarium at Las Animas, Colo., and during this period gave rise to a greater number of discharges for physical disability than any other one disease. Among the predisposing causes on board ship are much confinement below decks, promiscuous expectoration, and the sailors' habit of sleeping upon a mat on the deck, where they are most likely to suffer contact with inspissated sputum. In the old navy of masts and spars, the complement of a vessel, practically to a man, was forced, by the character of their duties, to spend much of the time in healthful outdoor employment, thus greatly counteracting the bad effects of the foul, ill-ventilated compartments below. In the modern fighting ship conditions are reversed, and we find but a small proportion of the crew performing duties in the open, and all the exercise accruing when sails were the motive power



is lost. To meet these changes and, really, to make them possible, extensive and carefully-planned systems of artificial ventilation, both of the supply and exhaust type, have been developed, until at the present time the deepest and innermost compartments are well supplied with fresh air, and some vessels are now fitted with combined heating and ventilating plants. But, in spite of these advances, in spite of limitation of dampness and expectoration, in spite of compulsory exercises in the open air and the stimulation to sports and athletics, in spite of every effort to eliminate at the recruiting station those predisposed, to detect and remove those in the incipient stages, and to keep the general health of all on a high plane, this disease persists, and statistical tables show that those whose duties confine them below decks furnish the highest rate per thousand. The table above referred to shows that the hospital corps men suffer more than any other occupational group. Efforts to eliminate incipient cases, both at the recruiting stations and in the service, have been doubled; isolation of cases is carefully observed, methods for more efficient ventilation are being worked out, provision for periodic disinfection of vessels throughout have been made, and circulars of instruction and rules regarding the conduct of antituberculous work have been issued from time to time.

As the result of political developments in the past few years, the navy personnel has been compelled to spend much of its time in tropical climates, both ashore and afloat, and as a result malaria, dengue, and dysentery have been brought into undue prominence. When afloat there is little danger, as the vessels lie far from shore and the food and water supplies are above reproach, but on shore, especially under the unfavorable conditions surrounding landing parties, military expeditions, etc., much damage results. This is well shown by the high rate of these diseases among the enlisted force of the Marine Corps, the group which performs the larger proportion of the duty on shore in the tropics.

Energetic efforts are being made in this country to diminish the incidence of lead poisoning among those whose work exposes them to the harmful effects of this metal, and the observations on this important question in the navy recently took concrete form in the following circular letter:

The Bureau desires to have the question of lead poisoning among the personnel on shipboard and at navy yards given careful investigation. It is, therefore, requested that particular care be taken to determine the existence of such cases, and that a full report be submitted in each instance, indicating the probable mode of lead absorption, symptoms, course, and termination, with recommendations regarding the adoption of appropriate preventive measures. Particular attention is invited to an article in the April issue of the *Naval Medical Bulletin*, by Medical Inspector E. R. Stitt, United States Navy, regarding lead poisoning through the inhalation of red lead-laden dust.

The following data are pertinent to the investigation of this subject.

Lead poisoning in persons in the navy may be brought about in the following ways:

1. Ingestion of lead as the result of taking food without properly washing the hands and face after painting.
2. By inhalation of lead-laden dust in compartments where lead dust is present as a result of chipping old lead paint, or otherwise. This dust may also be swallowed in the saliva, or at the time of eating or drinking.
3. By solution in the sweat of red or white lead deposited on the skin and subsequent absorption through the skin. Lead absorption by this method is of slight practical importance.
4. Methods of poisoning by lead through drinking water, or in coloring matter in food, are of less importance in the naval service than in civil life.

There are four chief types of lead poisoning: (1) Lead colic; (2) lead encephalopathy, in which the central nervous system is prominently affected; (3) the peripheral neuritis type, as shown in "wrist-drop"; and (4) lead cachexia.

If the period during which the lead poisoning has taken place be short, the manifestations are chiefly those of colic, or lead encephalopathy. In the diagnosis of lead poisoning, the blue line on the gums is the most constant and most easily observed sign. The value of this sign is chiefly negative, as probably more than one-half of those working in lead—but not having lead poisoning—show it. The typical blue line is on the gums, commencing at the tooth margin. Associated with this lead line, in cases of poisoning, will probably be found anæmia, loss of appetite, metallic taste, foul breath, and spots before the eyes. Headache is often a prominent feature of lead poisoning. A tendency to nausea, with a sense of weariness, is suggestive.

With lead colic, the pain tends to centre at the navel, but may be complained of in the region of the appendix. The patients are very restless and tend to roll about. The pulse is slow, small, and of high tension. Constipation is the rule, and its relief may relieve the colic.

In lead neuritis, the wrist-drop is usually preceded by pain, or by numbness of the muscles of the forearm. It is usually bilateral, but the extent of the involvement on the two sides varies. In lead encephalopathy, epileptiform seizures are the most common manifestations. Workers in lead who give absolutely no signs of lead poisoning may show as much as three parts per million of lead in the urine. This finding, like the lead line, is more confirmatory than diagnostic. A simple test for lead in the urine is that described by Von Jaksch: Place a strip of bright magnesium ribbon in about 300 c.c. of urine, and add 2 grammes of ammonium oxalate. After 2 to 12 hours, remove the magnesium strip, wash in

distilled water, and dry it. Next warm the strip with a crystal of iodine in a watch glass over a low flame. The formation of the yellow iodide shows that lead has been deposited on the magnesium strip. It will show one part in 50,000 of urine. (Considerable experience with this test is necessary for a proper interpretation of the findings.) A more satisfactory test is to dissolve the magnesium strip with the lead deposit in a very few drops of nitric acid; then make alkaline with soda solution; next add acetic acid, drop by drop, until the solution is perfectly clear; finally, add a few drops of a solution of potassium chromate, or bichromate, and note the formation of a yellow precipitate of lead chromate. For the detection of minute quantities of lead, more exact methods are necessary, involving concentration, destruction of organic matter, and electrolysis. Such methods, however, are difficult of application.

As regards the value of punctate basophilia in the diagnosis of lead poisoning, Sir Thomas Oliver considers it of value, but unreliable, having found the sign negative in 40 per cent. of his cases of plumbism. For this test, make a uniform smear on a slide and stain according to the Giemsa or Wright method. Blue dots in the red cells show punctate basophilia. The blue dots in lead poisoning seem larger and more splotchy than those usually observed in secondary anæmias from malaria or infection with animal parasites.

Prophylaxis: Men working in compartments containing lead-laden dust should wear some type of respiratory mask, and care should be taken that they do not become fatigued, a condition which predisposes to lead poisoning. Alcoholics are particularly susceptible to lead poisoning, and in particular to lead encephalopathy. In all forms of employment with lead a full meal should always be taken before commencing work. Thorough washing of hands and face before eating should be insisted upon, as well as bathing of entire body daily. Chewing of tobacco while working with lead should be prohibited. The taking of tablets of sodium hyposulphite is to be recommended during the continuance of work with lead. About 5 five-gramme tablets may be taken daily. The wearing of gloves during the process of handling, grinding, or mixing lead paints is of great value in preventing poisoning. Clothing that is tight-fitting at wrists and neck affords additional protection; cuts and abrasions should be protected until healed. When a workman complains of symptoms of lead poisoning, he should be directed to report to the surgeon for observation, and should the diagnosis be affirmative he should be removed from further contact with the source of poisoning.

As regards treatment, it should always be borne in mind that potassium iodide should be used with caution in cases of acute poisoning, as lead deposited in the tissues may be re-dissolved and intensify the existing condition.

Practically throughout the service, paints are received ready-mixed, including the red lead used so universally for a protective coating on metals, and the dangers associated with the handling of lead in the dried state (grinding, mixing, etc.) are confined to the civilian employees at the large naval stations where this work is carried on. The navy personnel properly apply great quantities of paint, and the necessity for care is, and has been, urged upon those ex-

posed. A fertile source of poisoning has been found to lie in the operation of "chipping" paint work; *e.g.*, removal of the old paint and accumulated rust beneath by the blows of a hammer. Under these conditions much of the paint is disseminated as a fine powder, which floats in the air and may be taken into the lungs with each inspiration. Cases originating in this way are particularly apt to occur in those working in the confined spaces below decks and, more particularly, in the double bottoms.

The percentage of flat-feet among the civilized races is very high, but in the usual vocations of our citizens it causes only a negligible amount of disability. In the navy, however, it presents a perplexing problem, involving, as it does, the initial physical examination of applicants, increased service activity, and malingering. It is worthy of note, in passing, that of all rejections by reason of physical unfitness, flat-foot furnishes a rate of 88.2 per thousand.

It is a simple matter to recognize and discard the developed case, but to eliminate the "weak-foot," to debar those who may give way under the stress of long and fatiguing hours of standing watch, requires the utmost care and judgment, and even then a considerable number of cases occur and require discharge from the service. The issue of properly-shaped and properly-fitting shoes, the inauguration of properly-gauged practice marches and special exercises, and critical examination and treatment when symptoms occur, have done much toward limiting the damage to the service from this source.

We must now consider that group of conditions, of absorbing interest, which results solely from the duties and environment peculiar to the naval service. First and foremost comes the subject of eye-strain, for it may well be said that the effectiveness of a naval fighting machine depends largely upon the endurance and acuity of the visual mechanism. Injury results from the use of telescopic sights on guns, and the periscope in submarines, from the glare of high-power searchlights, from the intense light and heat of ship furnaces, and from the necessity for the constant use of artificial illumination below decks.

The individual who is accepted for the service may be considered as having good vision and to be free from ocular disease, as the prescribed tests, properly applied, will eliminate those who are below par. From this picked body the gun pointers are selected, after an



unusually searching examination, and only when it is demonstrated that they can quickly and correctly read the fifteen-foot Snellen type at twenty feet or more. Even upon these eyes of exceptionally acute vision the use of the telescopic gun-sight is a heavy strain, and they are constantly watched for any sign of incipient trouble, and receive the most painstaking care and consideration. The vibration and motion of the large battleship, which make it so difficult and such an effort to use continuously these sights of great magnifying power, are present to a much greater extent in the submarine, and the use of the periscope in vessels of this class results in a correspondingly greater strain and requires, therefore, special effort in the direction of observation and treatment. Thus far it has not been found possible to do away with the strain resulting from the use of these two forms of optical instrument. All that can be done is to select the best eyes that can be found to start with, protect them from injury so far as possible, observe them constantly for signs of deterioration, and apply necessary treatment promptly and thoroughly.

The eye troubles resulting from the use of searchlights have been largely overcome by colored glasses, and all who handle this potent source of injury are required to wear them. In the fireroom, however, it is not so simple a matter. Here, in addition to the intense light, there is a blistering heat and often considerable moisture, mingled with draughts of cold air. This gives rise to "fogging" of the glasses, necessitating their removal. If an effort is made at the same time to protect the eyes against flying cinders and coal dust by using goggles, as is the habit with automobilists, the "fogging" or "steaming" resulting is much more marked. A satisfactory method of protection has not been devised, and it would appear that the only solution to the problem is the substitution of oil for coal as fuel. With oil there is no need for constant opening of furnace doors, the flame can be observed through colored glass at the side, and there are no flying cinders and no coaldust-laden air.

The important subject of artificial illumination on board ship can only be touched upon in as brief a paper as this. The problem is rendered particularly difficult owing to the peculiar structural conditions existing in vessels designed primarily for fighting, resulting, as it does, in little head room between decks, limited bulkhead space, and many obstructions. This leads to the necessity of placing the



sources of light low, practically on a line with the eye, and when it is realized that a considerable portion of the interior of a ship has to depend entirely upon artificial illumination, the bad effects upon the eyes can readily be appreciated. The whole subject of illumination of vessels will be taken up by a special board in the near future, with a view to rectifying these conditions so far as possible. Much has already been done to establish proper location of lights for desk work and reading, to replace the universal white with a neutral tint for inside decoration, and to substitute an unglazed, tinted paper for that which is smooth, white, and shiny.

The harm resulting to the ears from gunfire has long been recognized, but until lately no determined effort had been made to reduce the damage. The injuries vary from simple "ringing in the ears" and rupture of the drum to permanent interference with the functions of the delicate nerves of the internal ear and total deafness; the severity of the lesion depending upon the relative position of the individual to the gun, the repetition of exposure, and the presence or absence of protecting devices.

Many believe that the condition of the ventilating apparatus of the middle ear has much to do with the results produced, stating that greater injury obtains when the eustachian tube is closed or obstructed than when freely open. As a matter of fact, observation has shown that in many cases the tympanum is without doubt ruptured by a force from within, evidently the result of a positive wave of pressure, delayed by passing up the tube, reaching the drum within, just as the negative phase of the blast is present in the external ear. It has been amply proven that those least injured are to be found in the turrets, while those who are in the open suffer the greatest harm, especially if they are in advance of the muzzle. The small calibre guns produce more harmful results than those of large calibre.

The most universally used protection is a plug of cotton, absorbent or non-absorbent, and this simple method, when properly applied, is most efficacious. It has been the custom to issue the material to each individual exposed and allow him to plug his own ears, but it has been found that many did this improperly, getting the plug either too loose or too tight, with resulting injury. Furthermore, in some cases the plug has been found driven in so far as to necessitate a head-mirror and special forceps for its removal. At present, many medical offi-

cers personally insert the plugs, and the freedom from injury in these cases well warrants the effort involved. Cotton worked up with putty has been found to make an excellent plug, and one which holds its position well. Of special ear-protecting devices several have received practical tests, the best known being the "Elliott Ear Protector" and the "Frank Ear Stopple," but none has proven entirely satisfactory, owing to the diversity of requirements—maximum of protection, ease of adjustment and removal, long life, minimum reduction in the passage of voice sounds, non-interference with the use of telephones and voice tubes, and universal applicability to ears of varying shape and size.

Although protection is the goal to be attained, the prompt and adequate treatment of injury produced is not lost sight of, and many ears have been preserved that would otherwise have been lost or permanently damaged. This is specially true of ruptured drums, which, if promptly recognized and appropriately treated, usually heal kindly. Since many ruptures take place without external evidence or the knowledge of the individual, it is becoming a custom to examine all who have been exposed in order to detect this injury early, prevent infection, and assist the process of repair.

Heat exhaustion and thermic myalgia are found mostly among the fireroom force, men who perform hard, manual labor, oftentimes in excessive temperatures and under the heightened atmospheric pressure associated with the production of forced draught. Although heat exhaustion is uncommon and thermic myalgia never fatal, the total interference with duty is considerable and has led to efforts for their reduction. Ventilation and reduction of temperature have been accomplished, thereby minimizing heat exhaustion, but these very provisions have apparently increased the incidence of thermic myalgia, owing largely to the establishment of cold draughts. Extensive observations have been made in fire- and engine-rooms during speed trials and endurance runs, and it has been conclusively demonstrated that the ventilation is excellent, relative humidity low, and, with few exceptions, the temperatures not excessive. The cases of heat exhaustion have been confined to those physically below par (alcoholics, "green" and "soft" men, and those suffering from venereal disease). The cases of thermic myalgia were confined to those who drank excessively of cold water and who, during their resting inter-

vals, stood in the direct draught from the ventilators; the element of physical unfitness likewise played a part.

In order to meet these conditions the men for this duty are selected by a higher physical standard, an effort is made to train them in their duties prior to endurance runs,—they are cautioned to keep out of the direct draughts from ventilators and are not allowed to drink cold water. It is the generally-accepted opinion that, when oatmeal or extract of ginger is added to the water consumed, the incidence of myalgia and exhaustion is reduced, and this is being done on many ships to-day.

The subject of caisson disease in the navy comes up in connection with diving, and, although there have been but few serious cases, many divers have exhibited mild symptoms. This has been found to be due solely to lack of care in coming to the surface, and it has been the endeavor to have all divers and their helpers instructed in the importance of stage decompression.

Accidents, as elsewhere, furnish a large portion of the annual damage rate. Many of these casualties are inseparable from life on board of a modern fighting vessel, with its cramped space, complicated mechanisms, high explosives, and the demand for quick and accurate action, in spite of every preventive and safety device. Nevertheless, careful investigation has demonstrated points where improvements can be made, and new inventions are continually coming to the front and being adopted to minimize these risks.

Among the accidents, drowning takes first place, and it is appalling to note that during the year ending June, 1911, this alone gave rise to 109 deaths. It stands at the head of the list of causes of death, pneumonia being second with only 35 to its credit. Investigation has shown that every precaution is taken for rescue; lifeboats are always ready and can be manned in a few moments, the most modern life buoys are always at hand and can be released instantly, and drills of "man overboard" are constantly carried out. It is evident that no stone has been left unturned from a rescue point of view, but the death roll persists, and not until recently was it borne in upon us that the fault lay largely with the individual who was to be rescued. The astounding fact was discovered that a very large proportion of the navy personnel could not swim, and many could not even keep their heads above water for a brief period. Here,

indeed, was a weak point, and, to strengthen it, general orders have been issued making the art of swimming compulsory. An individual must now remain under instruction until he is capable of attaining a certain proficiency; swimming exercises, whenever opportunity offers, are no longer voluntary, and the diplomas of three midshipmen who should have graduated from the Naval Academy this spring [1912] were withheld by reason of their failure to attain the required standard in this branch of their physical training. In connection with this subject it is interesting to note the remark (which appeared in a recent volume) of the surgeon of one of the Japanese ships sunk by a mine, to the effect that only those were saved who could swim.

It is fully realized that this alone will not eliminate death by drowning, but it will reduce it to a marked degree, and it is hoped that by the introduction of the pulmotor, which is now being given practical tests, there will be a still further saving of life.

Another source of injury and death on board our ships is the explosion of gunpowder, the bursting of steam pipes and boiler tubes, and the effects of poisonous gases. These accidents are very uncommon, owing to the fact that every precaution is taken to prevent them; still, they do occasionally happen, and their effects are greatly intensified by the confined spaces in which they occur and the inability of rescue parties to render assistance. It is morally certain that in accidents of this class, if there were some way of reaching the victims early, whether in turret, fireroom, or bunker, many lives could be saved, and, with a view to accomplishing this end, various types of "rescue apparatus" are being investigated and given practical tests. Under these circumstances, as in the treatment of the apparently drowned, the pulmotor would no doubt prove of inestimable value.

In time of peace the question of dealing with the poisonous fumes resulting from gunfire is not of pressing importance, but in battle, when the vessel is wrapped in these noxious gases, grave problems relative to ventilation of the deeper parts of the vessel are developed. This matter, however, is best left to discussion under naval surgery, as is the case in preparations for the handling of the wounded, the location of battle dressing stations, provisions for evacuation of the wounded, etc.

The personnel of the navy, it must be realized, is composed of

picked men, physically and mentally, who live and work amid complicated mechanical and electrical appliances, in cramped spaces, many of which never see the light of day. They are exposed to every variety of weather and sudden change of climate. Their duties are arduous and exacting, and include many vocations in which civilians engage and, in addition, those of a military establishment. They are really a highly specialized body, and it is the duty of the medical officer to maintain that standard of physical condition which will preserve these trained units to the service and keep them in the highest possible state of efficiency.



## INDUSTRIAL POISONING

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THE subject of industrial poisoning by metallic and other chemical substances has as yet attracted little attention in America, while on the continent of Europe and in England the matter of the numerous deaths and sicknesses due to poisoning resulting from the nature of employment has been considered with great care, and legislation has been accomplished which has resulted in a great diminution of such poisoning. Most of the sickness due to industrial poisoning is preventable, provided the employer makes certain improvements in the processes of manufacture, and will lessen, as far as possible, the amount of dust allowed to fly about in the factory; and provided, also, that the employee will take certain simple precautions as to cleanliness, such as not eating in dusty places, or with unwashed hands; the wearing of simple respirators, or the use of safety devices. One difficulty in the problem of prevention is that in many industries in which lead is used, such as smelting, etc., the men employed, being ignorant, often not understanding English, do not know that the dust they are inhaling is poisonous; and could not read warnings, even if they were posted; and the foremen and employers do not always take the precaution of warning these ignorant ones of their danger. In many cases, however, where employers try to make the work less dusty, by care in handling the products, by the use of sprinkling, and in other ways, it is found that the employees are not willing to use the necessary precautions—they are not careful about washing before eating, and sit down to eat in places where the poisonous dust has accumulated, instead of eating outside the works.

The most frequent form of metallic poisoning is that with lead, since there are 125 or more trades in which this substance is used in

some way which makes it dangerous to the operative. Besides lead, there occurs poisoning from arsenic, antimony, chromic acid, phosphorus, mercury, hydrofluoric acid, and naphtha.

Lead oxide or white lead, a compound of lead used in all kinds of paints, and in a large number of trades, is responsible for the largest amount of lead poisoning, much of the disease being due to the dust arising in its manufacture or to sandpapering coats of paint. This is seen in ordinary indoor house-painting, in carriage painting, in the manufacture of enamelled bath-tubs and fixtures, in which many coats are applied and smoothed with sandpaper, in decorating of railroad cars, and the like, and numerous other occupations in which lead in some way or other is used, such as the smelting of lead where the fumes and dust carried out of the flues are very poisonous, and the effects are seen on vegetation, on poultry, and on cattle in the neighborhood. This dust contains 30 per cent. of lead in its metallic form, and a variable proportion of arsenic and sulphur. Zinc ore also contains lead, and is dangerous in the smelting process.

The manufacture of the carbonate of lead, red lead or litharge, is another source of lead poisoning. The glaze which is applied to the product in the manufacture of earthenware and china also contains a variable amount of lead; in cutting files the file is placed on a lead bed, the metal from which becomes pulverized and is deposited all about the operative, and flies about in the air he breathes. In the manufacture of storage batteries red lead is rubbed into wire gauze, and the part of the lead that is not adherent to the mesh may become detached and float about. A considerable amount of lead dust is also inhaled in printing, linotyping, and type-founding, as well as in the handling of type in typesetting. Diamond cutting and the setting of precious stones is another source of poisoning, the gems being placed in a lead bed before they are cut, and particles of lead from the bed are detached in the process of cutting and polishing. Plumbers and makers of lead pipes are subject to lead poisoning, as the putty contains red or white lead, and is poisonous to the mixers; in dyeing yellows the substance used is chromate of lead; tinning hollow ware exposes to lead poison through the lead in the coating, etc.

In the Bulletin of the Bureau of Labor, No. 95, Dr. Alice Hamilton gives a most valuable account of the white-lead industry, which she investigated for the United States Government. She describes at length all the processes of manufacture of white lead, the causes which produce the dangerous dust, and the improvements that can be made in the processes of manufacture, such as have already been put in use in England and on the Continent.

The lead enters the system in these various employments, by being absorbed by the stomach, the respiratory organs, and, in small amounts, by the skin. The harm that comes from the presence of lead on the skin arises from not washing before eating, and from chewing tobacco which, while in the pocket, or by the dirty hand, has been impregnated with lead, and not from absorption through the skin. Many workmen and foremen are impressed with the idea that chewing tobacco is a protection against lead poisoning, whereas it is really a cause of the ingestion of lead. The use of alcohol in any form is injurious to the lead worker, since it predisposes to the effects of lead, especially on the nervous system. The dust inhaled by the respiratory organs may penetrate into the circulation, after exerting an irritating action on the lung tissues, and there are emanations from heated lead and from ordinary paint in the process of drying which are extremely poisonous when used in a confined space. Lead is swallowed in considerable quantities with the food, when the hands are not washed before eating; the clothing worn when at work also contains a large amount of lead dust, which is carried home and spread about the house, and thus the operative lives in lead by day and by night. Since the lead is absorbed slowly and in very small amounts, when poisoning first manifests itself there is generally a considerable amount of the poison circulating in the blood, and it is eliminated very slowly.

Lead poisoning may be acute or chronic. In the acute form the symptoms are severe colic, with constipation, anæmia, and feebleness. Colic may occur, frequently followed by more severe symptoms connected with the brain and the nervous system. The commonest form of paralysis seen is paralysis of the extensors of the hands and wrist, constituting "wrist-drop"; it may also involve the muscles of the legs, causing "foot-drop," and the muscles of the shoulders, fore-arm,

and trunk, and, finally, epilepsy, tremor, and hysteria, sensory troubles, and a form of encephalopathy which is often rapidly fatal may also occur. Lead affects the kidneys, through which it is eliminated, in the form of contracted kidney; it causes gouty symptoms, and arteriosclerosis; and through the lowering of the general health it predisposes to all sorts of infections, such as tuberculosis, erysipelas, etc. The encephalopathy may be in the rapidly fatal form of acute meningo-encephalitis, or it may appear as a chronic pseudo-paralysis. It attacks the optic nerve and causes neuroretinitis and permanent blindness from atrophy. It causes sexual paralysis in both male and female workers. In women it causes abortion, and this may occur even when the father only works in lead. The offspring of lead-poisoned fathers are four times more subject to convulsions than other children, and they may be idiots, epileptics, imbeciles, or degenerates, as a result of the parents working in lead. There appear to be personal and family idiosyncrasies to lead which are increased by the use of alcohol, poor food, and bad hygiene in causing poisoning. The lead worker shows a peculiar pallor even when there are no active symptoms. The blue line of lead deposited on the edge of the gums is a characteristic sign of absorption of lead, which will always aid in the diagnosis. It consists of lead sulphate deposited in the deeper layers of the epidermis; the sulphur coming from the food. Lead lessens the number of red corpuscles in the blood and the amount of hæmoglobin in the corpuscles, by poisoning the blood-making organs. Females are peculiarly susceptible to lead poisoning, and Oliver attributes this to the fact that the blood lost in menstruation causes the lessening of corpuscles.

In lead encephalopathy the affection may begin with an apparently trifling hysteria, and yet the patient may be dead in a few days. There may be acute headache, convulsions, coma, and death after a few hours or days. Lead poisoning may cause paresis, tabes, or insanity; it affects both the cerebral centres and the peripheral nerves. It causes premature senility, and men of forty have the feebleness and the appearance of men of eighty years, and the condition of quite young men and women is pitiable indeed. The man permanently paralyzed with "wrist-drop" can neither feed nor dress himself, his hands being practically useless. He may be so generally paralyzed as to be a helpless cripple for life, or he may be permanently blind;

nor are these bad effects always the result of a long period of working in lead, but men fresh from another employment, and not warned of their danger, may be attacked in from two to three weeks after beginning work.

The treatment of lead poisoning consists in the endeavor to eliminate the poison by magnesium sulphate, the use of potassium iodide as an alterative, and general supporting measures; mild purgatives locally applied and heat are useful for the colic; for the arm paralysis, massage, electricity, and splints to support the wrists may be employed; but recovery is very slow and may be only partial.

Prevention of lead poisoning, as of all metallic poisoning, lies in better ventilation of the works where it is handled, by means of exhaust fans which remove the dust-laden air; the changing of the process of production so that there shall be as little dust as possible, and the use of sprinklers in all dusty processes; the building of factories with hard, smooth floors and no places where dust can lodge; the use of wet sweeping whenever cleaning is done; absolute cleanliness in the care of the works, and the providing of proper lavatories. Rules have been made in England requiring the employees to take a full bath and to change their clothing before leaving the works, the time for the bath being given at the expense of the company. Lavatories with shower baths, hot as well as cold water, since cold water alone will not remove the lead, towels and soap should be provided. The workman should be given cap and clothing at the expense of the company, and should be required to change these before going home. In dusty places he should be required to wear a simple respirator furnished by the company, an attractive lunch-room where coffee and milk are served and food can be heated should be supplied. The education of the workman as to his danger should be a matter of concern to the employer, and he should have a company physician examine the workmen at regular and short intervals, and who has the power to send them off duty when he considers it necessary. It is needless to say that the company should continue to pay the victims' wages in such cases. Clothing for use in the mill should be supplied and laundered by the employer.

It is ignorance on the part of the employer and the employee that is at the root of the trouble in metallic poisoning. The employer is generally as willing to take precautions, when he is informed of the



dangers to the workman, as the employee when he learns of his danger.

An important point in the prevention of paint lead poisoning is the use of a substitute for lead. In paint for inside use the substitution of non-poisonous zinc white or zinc oxide for white lead has been adopted in Europe, and it has been found as good as lead for all interior decorations; it is not quite as durable as lead for outside work. Lead-sulphate, being insoluble, is also not dangerous. Belgium, Germany, England, and Holland forbid dry sandpapering, and require the work to be done wet, thus reducing the amount of dust. France uses only zinc white, while in other countries the amount of lead that may be used in the paint is limited. Germany compels employers to furnish proper washing places with hot water, and forbids the chewing of tobacco. Among printers, one of the principal elements in lead poisoning is the dark, unsanitary places in which the men work, predisposing to tuberculosis; and plenty of light and sunshine in the workrooms and the enforcement of sanitary precautions would go far to prevent this disease so prevalent among printers and typesetters.

Mechanical artists who prepare colored pictures for catalogues and other advertising material used in business suffer from lead poisoning due to sucking the small brushes used in order to bring them to a point, before dipping them in the paint, not thinking that the paint contains lead. They also use a brush that sprays the paint on by compressed air, and so makes a cloud of small droplets which is especially liable to cause poisoning. This spray spreads through the air and is inhaled by the workers. Employers, knowing that they supply lead paints, deliberately tell their men that there is no lead in the paint, otherwise, they claim, they could not get good workmen.

It is a matter of fact that the diagnosis of lead poisoning is not always made by the physician in attendance on the victim. Lead affects the system so generally, and simulates so many general and local diseases, that, unless the physician is on his guard and gives due weight to the occupation of his patient, he is not led to seek for poisoning as a cause, and so his treatment is not beneficial. It is also a fact that few physicians have studied the subject of industrial poisoning sufficiently to know in which trades lead is used. They know in a general way that painters are subject to lead poisoning, but

may not realize that the making of storage batteries or enamelled bath tubs, the smelting of zinc, the preparation and polishing of brass, the trade of the mechanical artist, the diamond cutter, and many other industries expose the workman to dangers from lead poisoning. It is due to this fact that cases of tuberculosis, caused primarily by the presence of lead which lowers the vitality of the subject, and only secondarily by the bacillus of tuberculosis, are treated in the ordinary way, while the important point to be gained in treatment is the elimination of lead from the system. Few persons realize that the abortions of a wife may be caused by the fact that her husband is a worker in lead, and not a syphilitic. Hysteria is generally attributed to an unstable nervous system or to lesions of the female genital organs, but hysteria in a lead worker may be the forerunner of acute encephalopathy, which will be fatal in a few days.

The oculist confronted with a case of neuroretinitis, or a case of nephritis, does not often look for lead poisoning as a cause of the eye symptoms. Thus it would seem advisable that our medical colleges should make a special study of lead and other metallic poisonings, and should see that every graduate is on the alert for such occupational diseases in his patients. America is the greatest manufacturing country in the world, yet it has made the smallest advances of any in the care for the health of its workmen. Immigrants are so many, and their services so cheap, that we have not yet learned, as have the older European nations from whom we draw our population, that to save the health and the lives of our workmen is an economic necessity. The profession has not yet learned that it would have to give less in charity practice if the working man and working woman were taught how to avert preventable diseases. Such work will not lessen their receipts, as has been suggested by some physicians, since machinery will still claim its victims, and bad eating will still remain to produce rheumatism and diathetic diseases. But such work will save the heartache that every right-minded physician must feel when he sees before him the worker of thirty or forty years of age, looking like a man of seventy, and effectually crippled for life by paralysis, blindness, or imbecility. The fact that lead poisoning is one cause of race suicide is well known to the investigator, but most general practitioners are not aware of it.

That the number of cases of lead poisoning is much more

considerable than is generally supposed is shown by the work of investigators. Dr. Alice Hamilton, in the investigation of 23 white lead factories, in which there were 1,600 men employed, found 388 cases of lead poisoning occurring in a period of 16 months, and these in all probability did not include nearly all the obscure cases. It is most difficult to get reliable statistics in this matter, since the employer does not know, or will not tell, of the cases that occur, and the investigator must depend on records of hospitals and dispensaries, and the information obtained from private doctors and from company physicians. In hospitals the lead cases are very often improperly diagnosed, and the searcher has to make a new diagnosis from his own knowledge of the symptoms of the disease.

*Brass* is a mixture of zinc, copper, tin, and lead, the amount of the latter being greater as the brass is poorer. Brass poisoning consists of a peculiar ague, which comes on after leaving work. The sufferer has chills, a dry throat, an irritating cough, and feeling of constriction in the chest, extreme prostration and lassitude, nausea, apathy, and shivering that is not relieved by increasing the coverings of the bed. This generally lasts an hour, and is followed by sweating and sleep. It is more common in winter on account of working with windows closed, and is caused by the inhalation of zinc fumes in melting the brass. There is a chronic form of bronchial irritation, and in the end the poison acts on the digestive organs, nervous system, kidneys, and liver, and hemorrhoids and constipation, with jaundice, are common. Symptoms of lead poisoning are seen in these same workmen where the amount of lead in the cheap brass is large. Brass polishers and finishers also suffer from lead dust rubbed off in the polishing processes.

Arsenic is used in mines, foundries, chemical and color works, in making of shot, in manufacturing glass, in printing and dyeing, in making wall-paper, oil-cloth, colored flowers and papers, colored chinks, in stuffing of animals, in tanning, and in etching of brass. It poisons the workman in the form of gas and dust, and may cause acute intoxication with gastro-intestinal symptoms in susceptible subjects. But the most common form of poisoning is the chronic cachexia produced by arsenic; ashen or yellowish color, bleeding of gums, gastro-intestinal symptoms, emaciation, loss of strength, falling of hair and

of nails, skin eruptions, irritation of the eyes and nasal mucosa, paralysis, violent cutting pains in the limbs, headache, sleeplessness, melancholia, epilepsy, and loss of memory all are present.

Mercury vapors are inhaled by workers who make incandescent lamps in which the vacuum is created by mercury, and by makers of X-ray apparatus, thermometers, and barometers. It is used in dyeing feathers and furs, and in making felt hats. It does not, however, very frequently cause poisoning. The symptoms supposed to be due to mercurial poisoning in hat factories are now believed to be caused by the steam and dampness.

Symptoms of poisoning are seen in dry-cleaners who use naphtha continually. After working for some time they become hysterical or stupid, and lose a taste for solid food, and a taste for stimulants results. They call this condition "naphtha-jag." Anæmia is an important symptom in old workers.

Hydrofluoric acid causes poisoning in the making of cut glass by the etching process; in the extracting of silicates from cane for wicker chairs; in the making of manure, and of superphosphites. It causes violent irritation of the mucous membranes of the eyes, nasal and respiratory organs, spasmodic cough, and ulcerations of the entrance of the nose, gums, and the mucous membranes of the mouth, and painful ulcers of the skin. It is inhaled as a vapor, or fine droplets of liquid suspended in the air of the workroom.

One of the most terrible forms of disease due to poisoning is phosphorus necrosis of the jaws, which occurs in match-makers. At the date of writing, Congress has just passed a taxation bill on matches made with phosphorus that will practically do away with white phosphorus matches in this country. France forbade the use of white phosphorus, the poisonous substance used in making matches, some years ago. There the industry is a government monopoly, and it was found that so much money was used in paying indemnities to workers that the profits were seriously lessened. The use of non-poisonous red phosphorus has taken the place of the white abroad. The fumes of the white phosphorus which are given off at an ordinary temperature cause an inflammation of the jaw, beginning about the roots of the teeth, especially if they be decayed, and extending to the bone of the jaw. These fumes are fatal to the life of the bone, which dying has to be removed. There are several instances recorded of workers

who have had to have one or both jaws removed, with terrible disfigurement and inability to chew solid food for life. Several of these patients were young and formerly good-looking girls. This disease is called by the operatives "phossy-jaw." Match heads are 5 per cent. phosphorus, and in mixing, dipping and forking the matches the fumes are inhaled. Methods of prevention have been used, care of the teeth, ventilation, etc. But the results have not been a removal of the disease, the only real preventive measure being to prohibit the use of white phosphorus. Another effect of phosphorus is a fragility of the long bones which occurs after years of its use, the patient often breaking a thigh from mere muscular exertion.

The prevention of all these forms of industrial poisoning is the same that has been outlined for lead in an earlier part of this article—practical methods of housekeeping and cleanliness in the manufacture of all products, rules made by the employer who understands the problem and enforced on the ignorant worker who does not know his danger; absolute cleanliness of the workman's hands before eating, and of his clothing before going home; the furnishing of proper lavatories, with hot water, soap, and towel, lunch-rooms with hot food at a small price, and the absolute prohibition of alcohol and tobacco, will go far to relieve the situation. Public health education used among the workmen, given in such a way as to interest him and help him, will do much more, and in this work the general practitioner must join and do his part with a will.



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